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Cologne, Germany — August 19-23, 1956

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DISEASES

of the

CHEST

OFFICIAL PUBLICATION

AMERICAN COLLEGE OF CHEST PHYSICIANS

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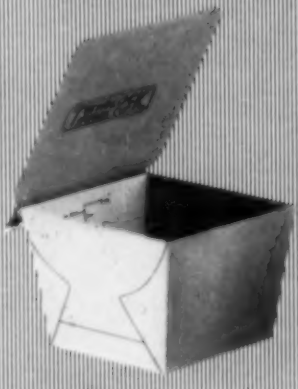

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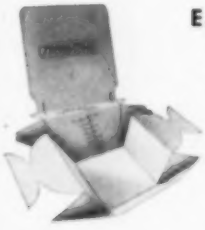

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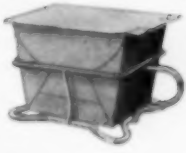

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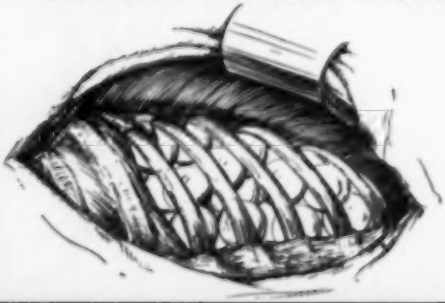

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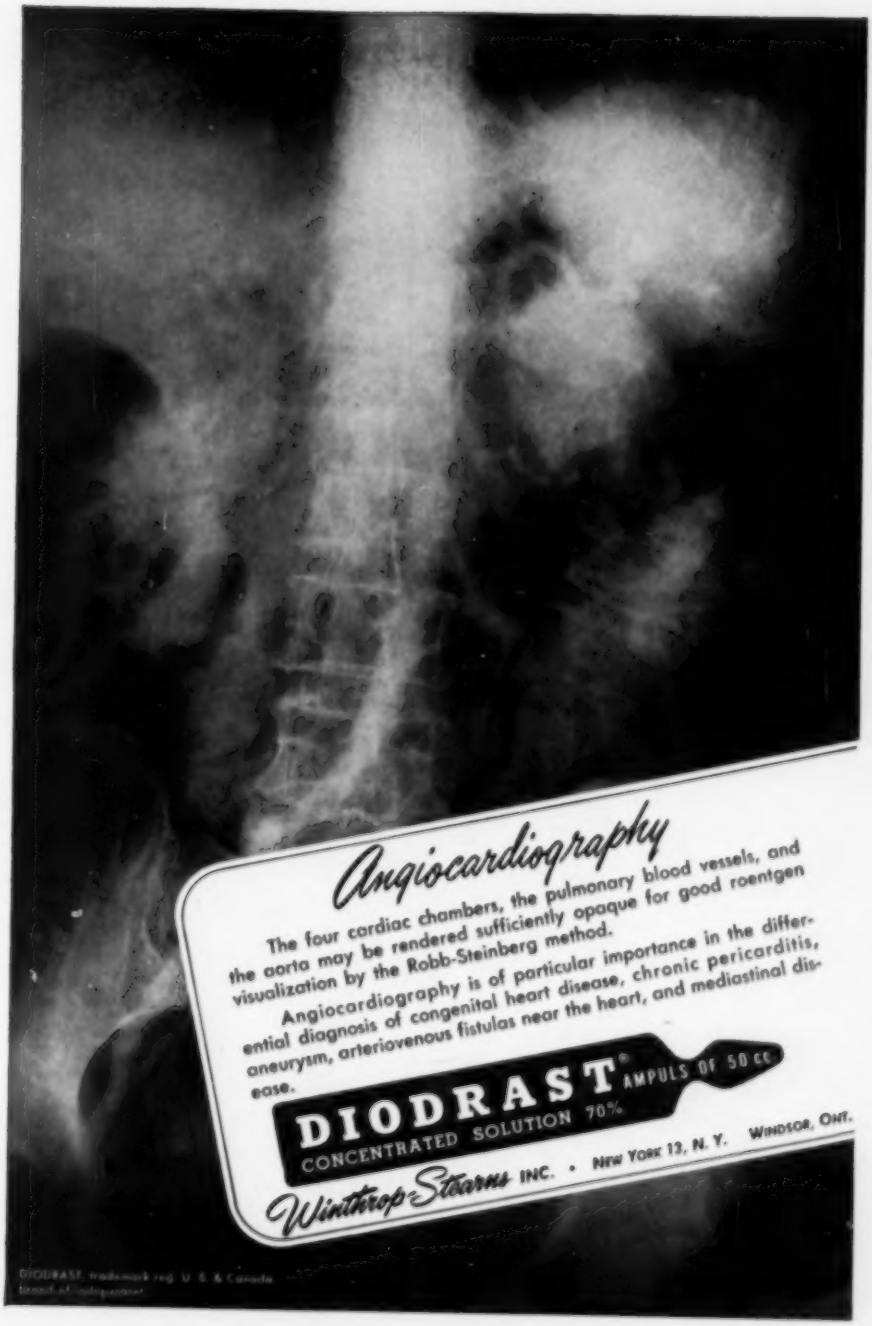
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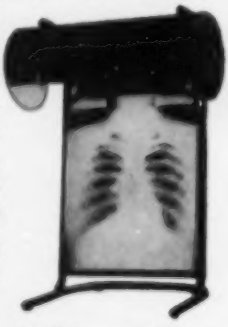

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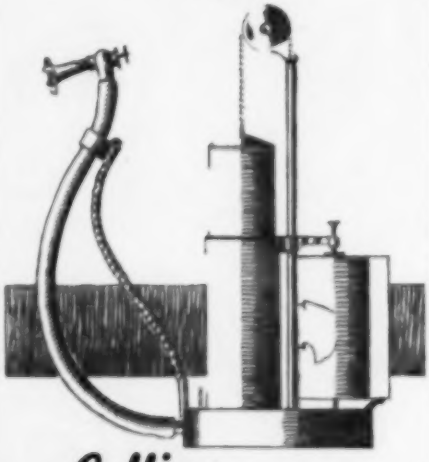

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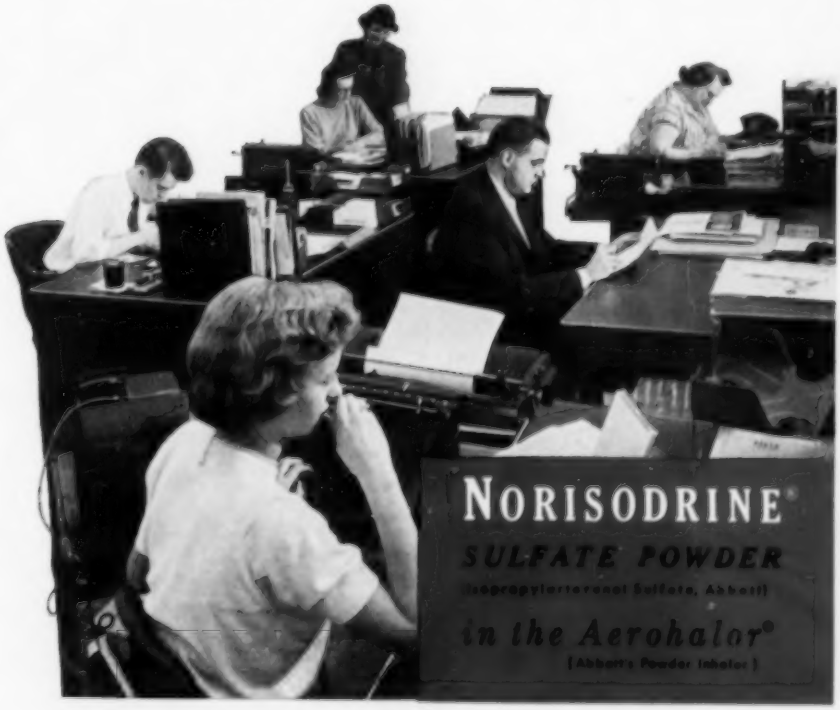

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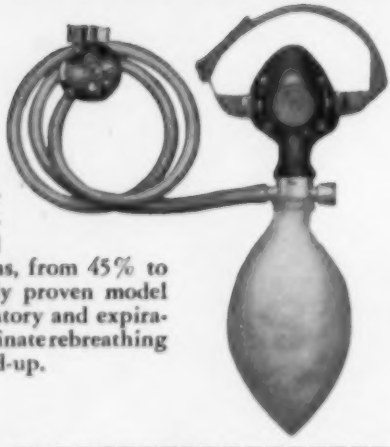

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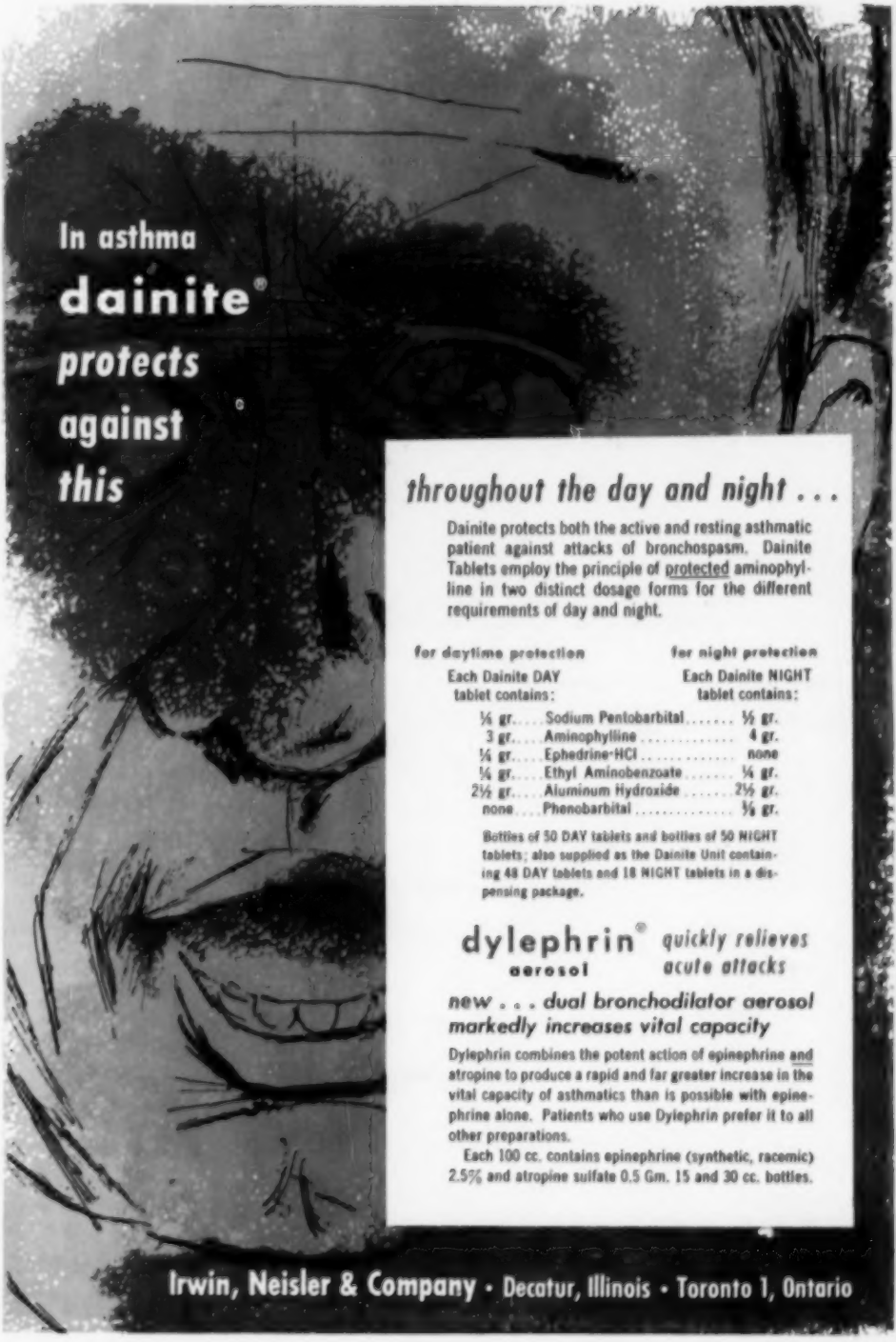

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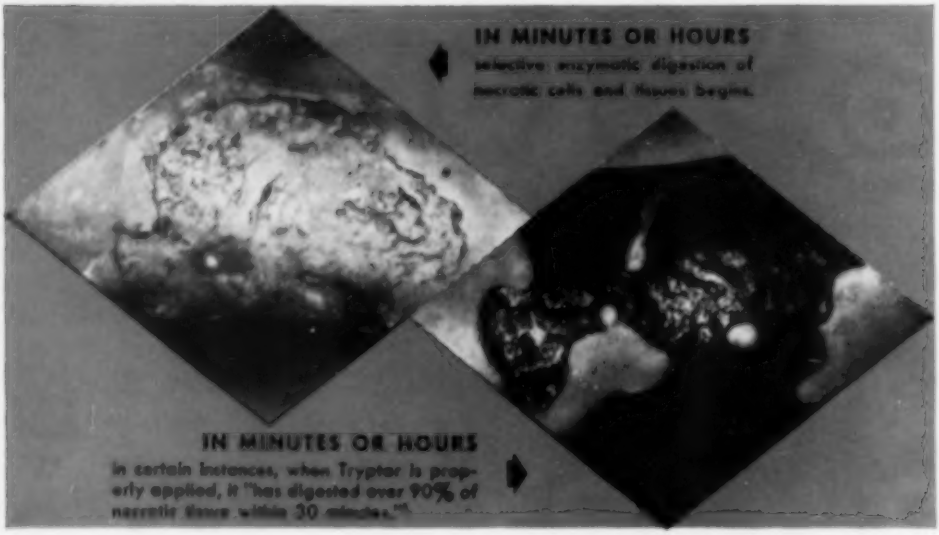
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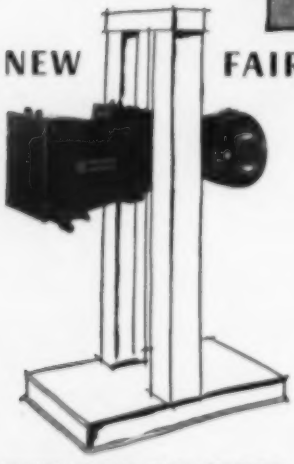

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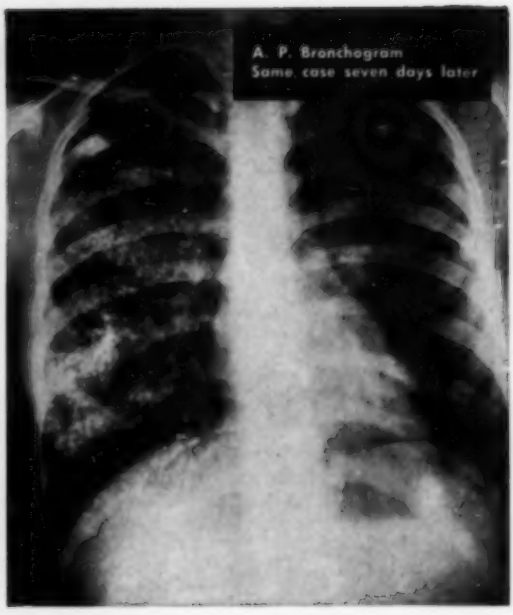

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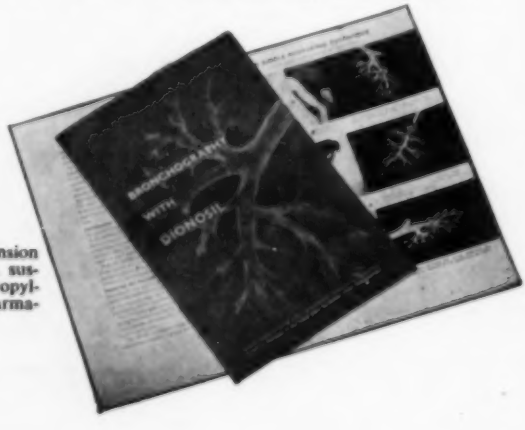

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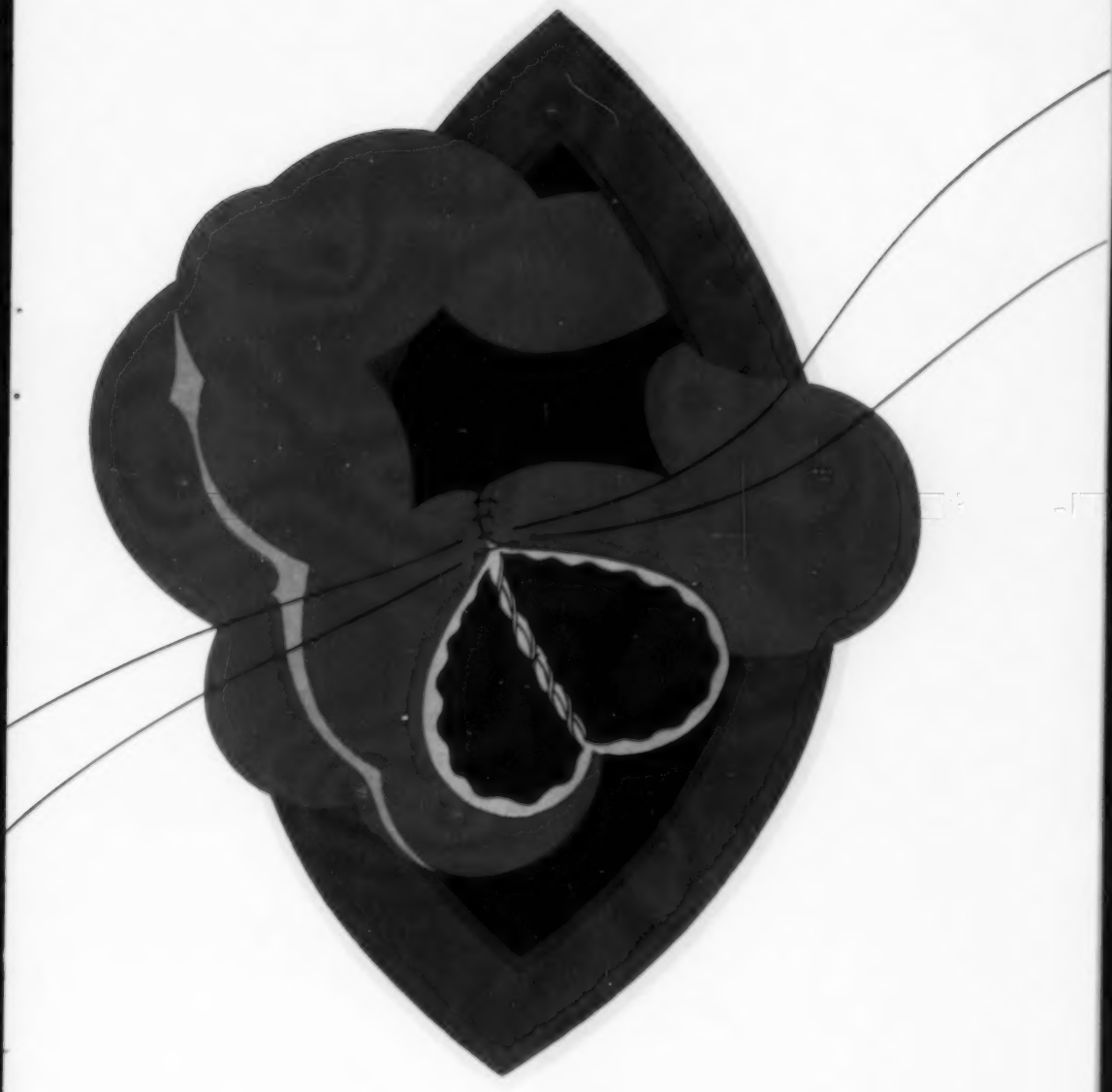

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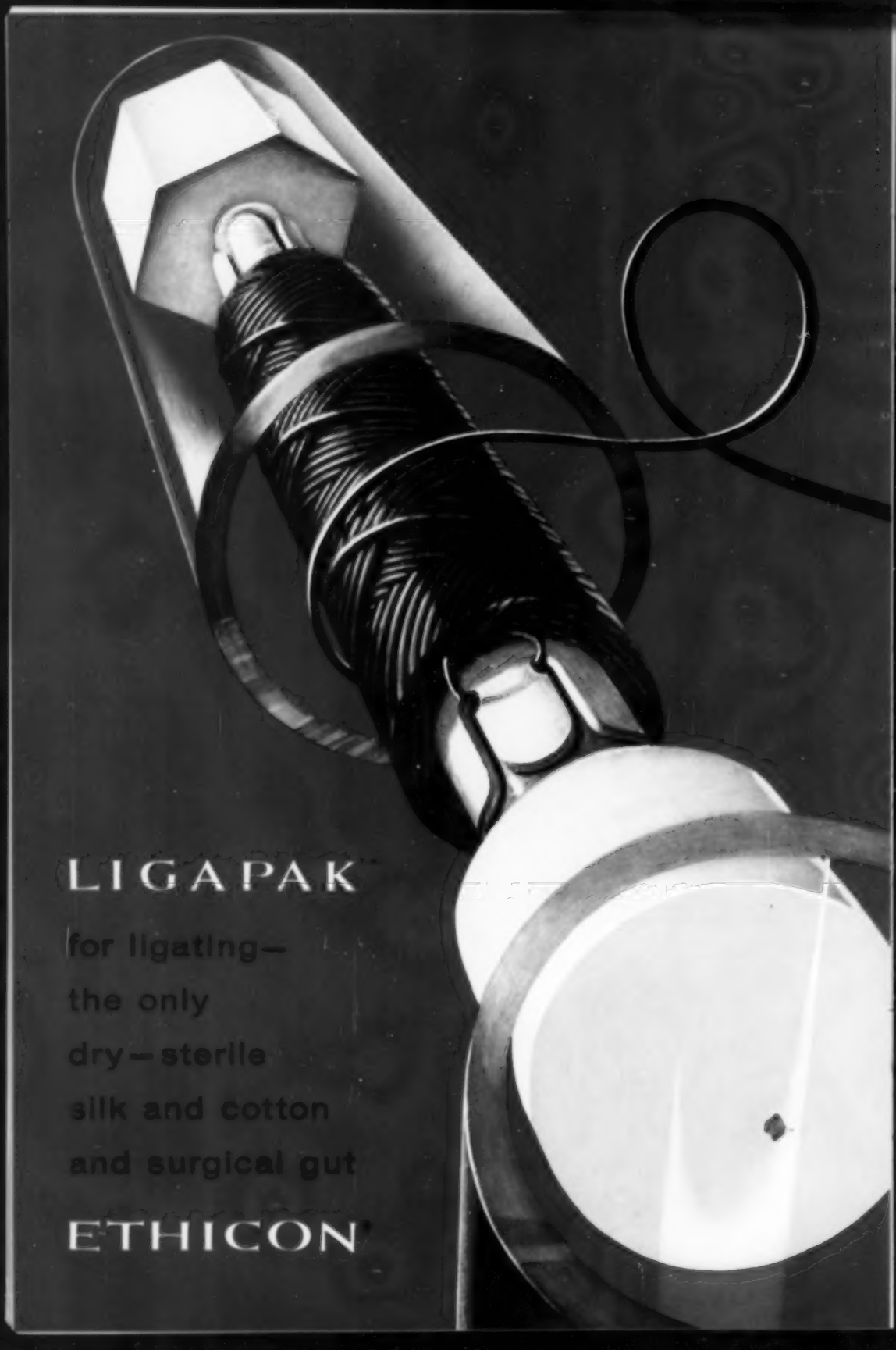
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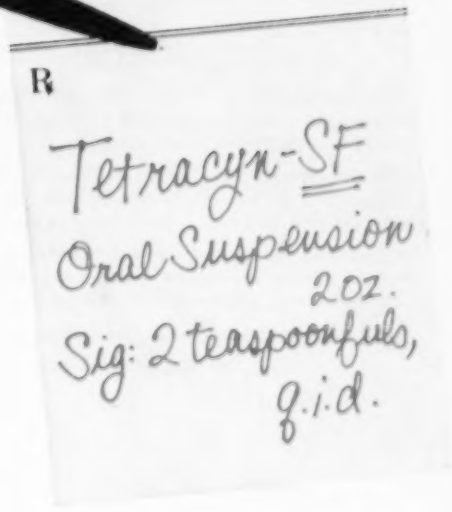

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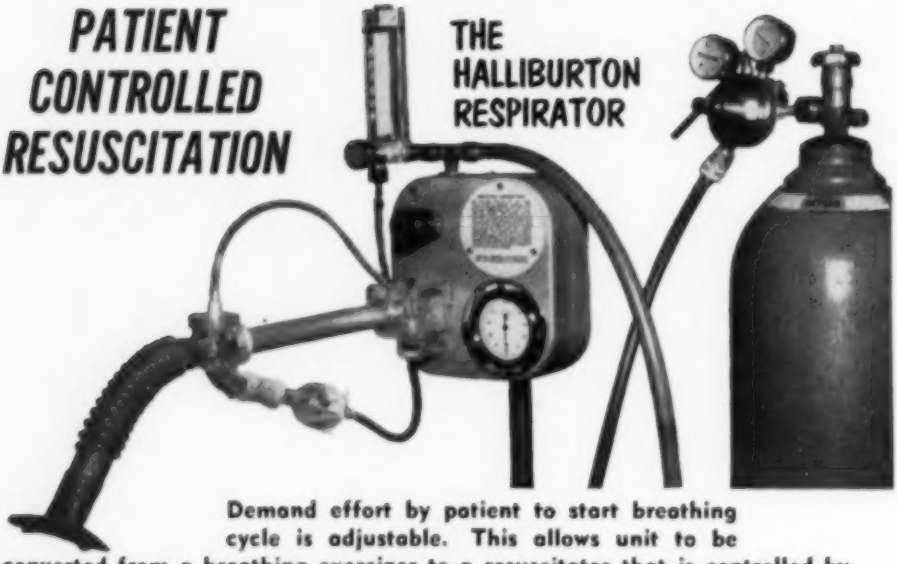

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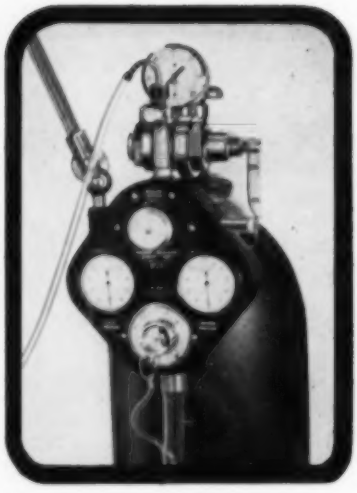

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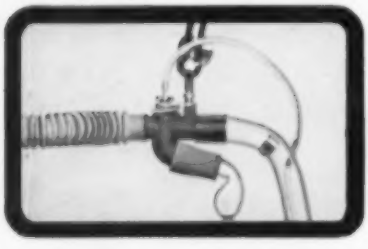

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DISEASES of the CHEST

VOLUME XXVII JUNE, 1955 NUMBER 6

The Tuberculosis Patient Who Leaves Against Medical Advice from the San Francisco City and County Hospital [1, 2]

HARRY A. WILMER, M.D., Ph.D.[3]

Palo Alto, California

A review of the literature fails to reveal a study in which any appreciable number of self-discharge patients is studied statistically in comparison with a control tuberculosis population. With this in mind, the self-discharge patients, i. e., those who go absent without leave (AWOL) or sign themselves out against medical advice, were studied for the year 1952 at the San Francisco City and County Hospital. This is a 1200-bed institution with 450 beds devoted to tuberculosis, half of which are under the medical direction of Stanford University and half under the medical direction of the University of California. In the year 1952 there were 524 routine discharge tuberculosis patients and 184 self-discharge patients. This report is from the Stanford University service.

Definition

The term *self-discharge* rather than the more usually employed "irregular discharge" is used throughout this paper, for it expresses what happens in terms of the individual rather than what it means to the hospital administration. The too often used word "recalcitrant," full of emotional meaning and moral judgment of bad or evil, is not used in this paper. In the writer's opinion it has no place in the classification of patient disorders; it cannot be defined so that different workers can agree as to its meaning. "A term," says Bridgman, "is defined when a condition is stated under which I may use the term and when I may infer from the use of the term by my neighbor that the same conditions prevail."

Sample Studied

For detailed study the case records of all self-discharge patients from the Stanford service were analyzed, numbering 74. A control sample of 74 routine discharge patients' charts was called from the Record Room, without selection and according to date of discharge, so as to equal the number of self-discharge patients in each calendar month. Five of the

1 Stanford University Tuberculosis Service, San Francisco City and County Hospital.
2 Aided by a grant from the Palo Alto Medical Research Foundation and by a grant from the San Francisco Tuberculosis Association.
3 Assistant Professor of Psychiatry, Stanford University, San Francisco.

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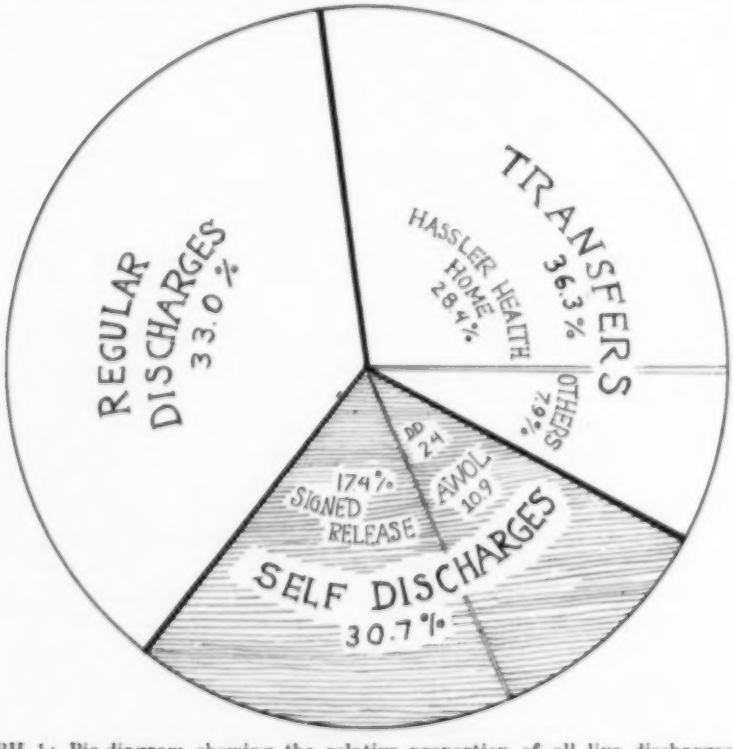
charts indexed as regular discharge were found on study to be self-discharge patients, an error of almost 10 per cent, so these were excluded, bringing the control portion to 69. In a few instances in the following tables the totals are not equal from table to table, because the information was not always available. The racial distribution was the same in the control and self-discharge samples: two-thirds white and one-third non-white.

TABLE I
Nature of Live Discharges, 1952

Discharge	No.	Per Cent
Routine Discharge	248	33.0
Signed Release	131	17.4
AWOL	82	10.9
Disciplinary Discharge	18	2.4
Transfers to Hassler Health Home	213	28.4
Other transfers	59	7.9
	751	100.0

Regular Discharge: 69.3 Per Cent
Self-Discharge: 30.7 Per Cent



GRAPH 1: Pie-diagram showing the relative proportion of all live discharges from San Francisco City and County Hospital in 1952. Self-discharges are divided into Signed Release, AWOL and Disciplinary Discharge.

It was found that 30.7 per cent of the patients fall into the self-discharge category. This group has three subdivisions: 17.4 per cent Signed Release; 10.9 per cent AWOL and 2.4 per cent Disciplinary Discharge (Table I Graph 1). Disciplinary discharges are included among the self-discharge patients because in essence that is what they are. The self-discharged constitute 33 per cent of live discharges at Hassler Health Home, to which the majority of transfers were sent; there was again a self-discharge rate of almost one-third the patient population.

If one divides the control and self-discharge into just two groups: far advanced and not far advanced disease, it is found that the majority of the control do not have far advanced, while the majority of the self-discharged do have far advanced disease. This is a significant difference: $Chi^2 = 4.3$, $P < .05$, or the probability of this occurring by chance is less than 5 in 100.

STATISTICS TABLE NO. 1

	Far Advanced	Not Far Advanced	Total
Control	30	39	69
Self-Discharge	45	29	74
Total	75	68	143

Sex

Although male admissions exceeded female admissions by 31 per cent, the female self-discharge exceeded the male by 3.8 per cent (Table II Graph 2). While this is not statistically significant, it is obvious from the figures that the relative problem of self-discharge is just as important in the female as in the male population. In the male group the patients

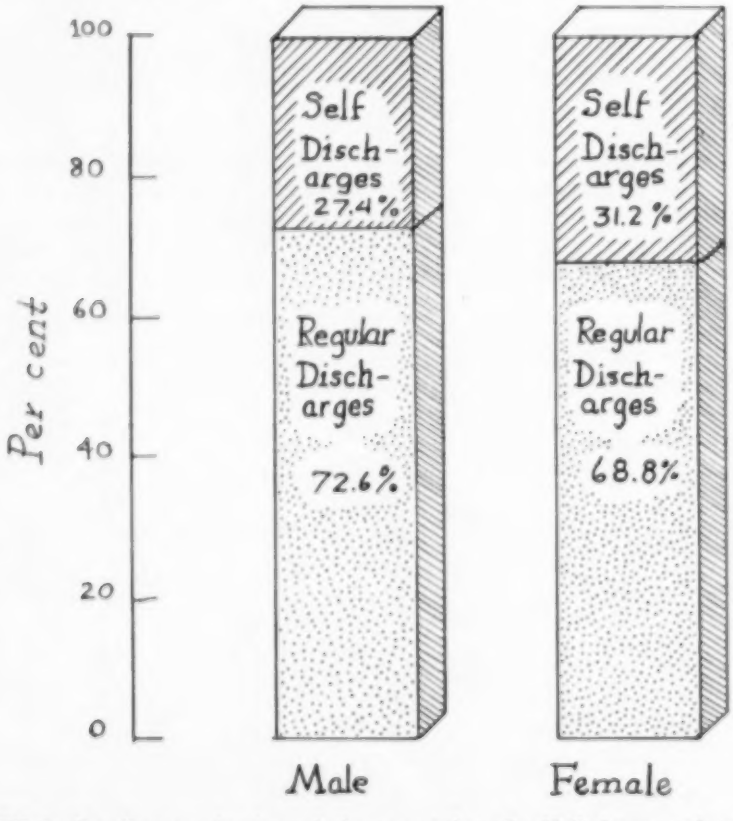
TABLE II
Self-Discharges for Entire Hospital, According to Sex

Discharge	Male		Female		Both Sexes	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Regular Discharge:						
Routine	167	44.6	106	70.7	273	52.1
Transfers	207	55.4	44	29.3	251	47.9
Total	374	100.0	150	100.0	524	100.0
Self-Discharge:						
Signed Release	75	53.2	54	79.4	129	61.7
AWOL & Disciplinary Discharge	66	46.8	14	20.6	80	38.3
Total	141	100.0	68	100.0	209	100.0
Totals:						
Regular Discharge	374	72.6	150	68.8	524	71.5
Self-Discharge	141	27.4	68	31.2	209	28.5
Total Live Discharges	515	100.0	218	100.0	733	100.0

going AWOL or by Signed Release are approximately equally divided. In the female group, however, about 80 per cent were Signed Release and 20 per cent AWOL. This is a significant difference: critical ratio — 2.6, P ⁄ .01, or such a difference occurring by chance is less than 1 in 100 that the females would exceed the males in this proportion in the Signed Releases.

It would seem that the women are less likely precipitously to leave the hospital without notification to their physicians, and this observation led to the realization that in a number of ways the AWOL group is a different one from the group who sign themselves out against medical advice. This is an important point, because to formulate programs to meet the self-discharge problem it is quite possible that a number of fallacies may creep into thinking if one does not divide and consider these two subgroups separately.

Of all the self-discharge patients, 39.6 per cent left with positive sputum, mostly on direct smear. Considering the contagiousness of disease in the Signed Releases, 63.3 per cent of the females had positive sputum com-



GRAPH 2: Bar-diagram showing relative proportion of self-discharge and regular-discharge, male and female.

STATISTICS TABLE NO. 2

Positive Sputum

	Male	Female	Total
Signed Release	21	19	40
AWOL	36	11	47
Total	57	30	87

TABLE III

Type of Self-Discharge According to Sex and Positive Sputum

Discharge	Male No.	Male Per Cent	Positive Sputum No.	Positive Sputum Per Cent	Female No.	Female Per Cent	Positive Sputum No.	Positive Sputum Per Cent	Both Sexes Pos. Sput. No.	Both Sexes Pos. Sput. Per Cent
Signed Release	70	45.2	21	34.4	60	80.0	19	63.3	40	44.0
AWOL	67	43.2	36	59.0	15	20.0	11	36.7	47	51.6
Disciplinary Discharge	18	11.6	4	6.6	0	0.0	0	0.0	4	4.4
Total	155	100.0	61	100.0	75	100.0	30	100.0	91	100.0

Percentage of Positive Sputum Among Self-Discharge Sample Where Data Available

	Signed Release No.	Signed Release Per Cent	AWOL No.	AWOL Per Cent	Disciplinary Discharge No.	Disciplinary Discharge Per Cent	All Self-Discharged No.	All Self-Discharged Per Cent
Positive	40	30.9	47	57.3	4	22.2	91	39.6
Negative	90	69.1	35	42.6	14	77.8	139	60.4
Total	130	100.0	82	100.0	18	100.0	230	100.0

pared to 34.4 per cent of the males. This is nearly reversed in the AWOL group, where the males with positive sputum predominate (Table III Graph 3). The reversal of the predominance of positive sputum of females in Signed Release and males in AWOL is a significant finding: $Chi^2 = 5.55$, $P < .02$, or the probability of this occurring by chance is less than 2 in 100.

History of Prior Self-Discharge

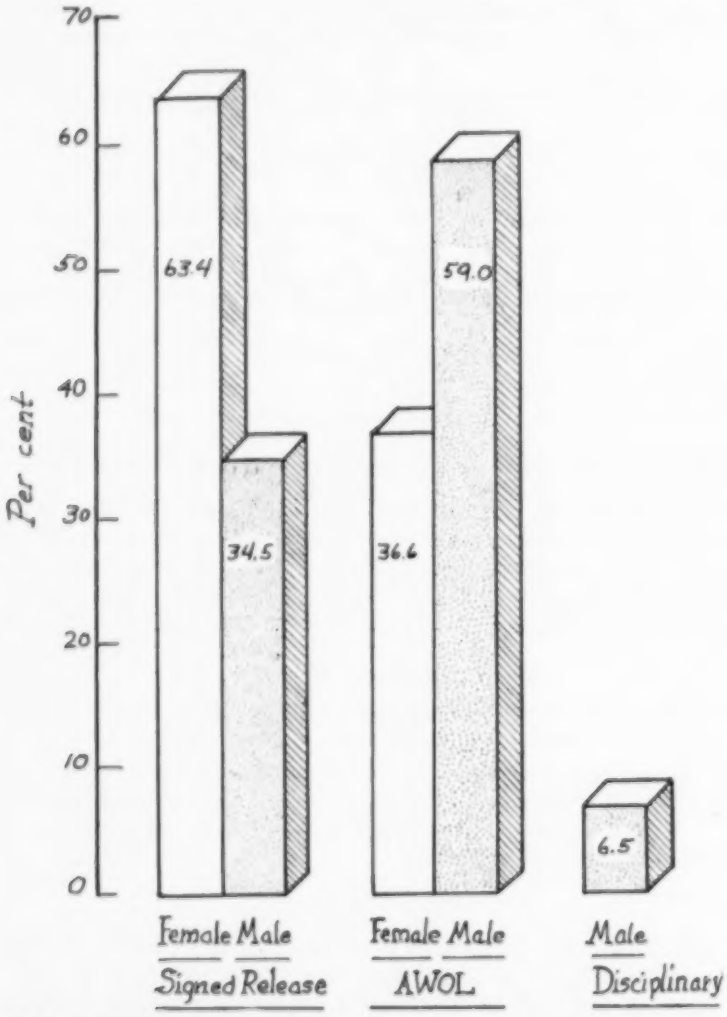
The self-discharge patients are multiple offenders. The whole self-discharge group showed a history of similar prior behavior in 37.1 per cent of the cases. There was a history of prior self-discharge in only 5.8 per cent of the control group. This is a highly significant finding: the critical ratio = 4.48, $P < .001$, or one would have to take 1,000 samples before finding this difference by chance.

Extent of Disease

It was found that patients who were readmitted to the hospital had more extensive disease than on their first admission. This was of course to be expected, but in Table IV Graph 4 the striking change in the number of minimal, moderately advanced and far advanced cases in the sample between first admission and readmission is shown. Considering the stage of illness in relation to first admission and readmission, it is found that

STATISTICS TABLE NO. 3

	First Admission	Readmission	Total
Minimal	138	43	181
Moderately Advanced	146	118	264
Far Advanced	194	218	412
Total	478	379	857



GRAPH 3: Bar-diagram showing the percentage of patients with positive sputum, according to type of self-discharge and sex. There are no female disciplinary discharges. The female Signed Release and the male AWOL predominate with positive sputum.

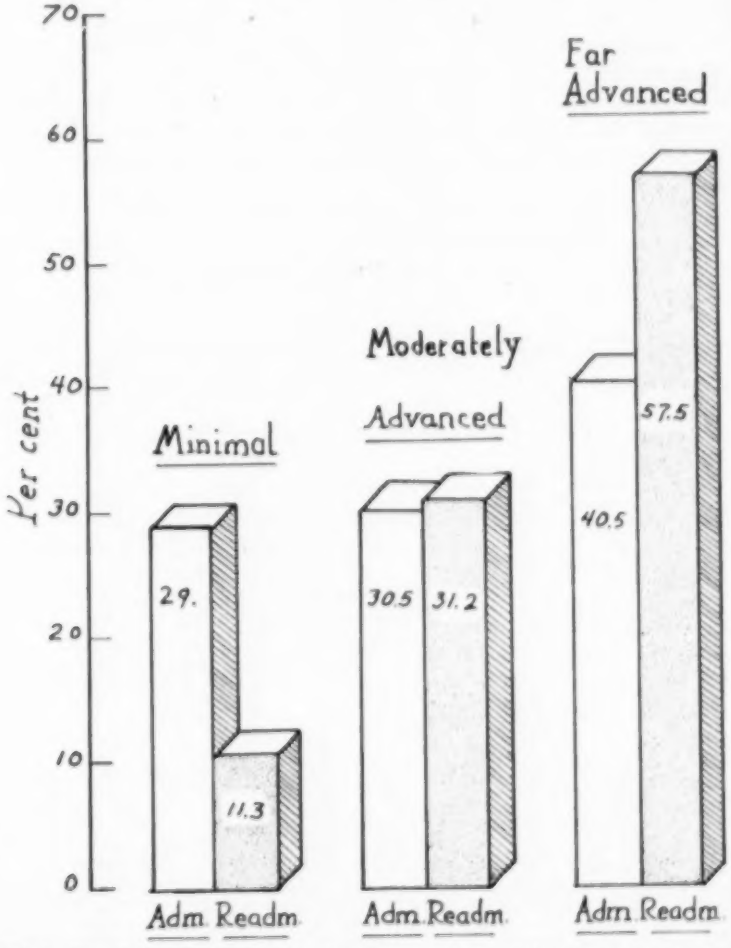
TABLE IV

All New Admissions and Readmissions According to Extent of Tuberculosis

Extent of Disease	First Admission*		Readmission**		Difference in Per Cent
	No.	Per Cent	No.	Per Cent	
Minimal	138	29.0	43	11.3	—17.7
Moderately advanced	146	30.5	118	31.2	0.7
Far advanced	194	40.5	218	57.5	17.0
Total	478	100.0	379	100.0	

*Males 69 Per Cent **Males 74 Per Cent

*Females: 31 Per Cent **Females: 26 Per Cent



GRAPH 4: Bar-diagram showing the breakdown of the status of disease in all newly admitted patients and all readmitted self-discharge patients for 1952. This shows the increasing severity of disease on readmission.

the minimals are more numerous on first admission, the moderately advanced slightly more numerous and the far advanced less numerous. The number of patients is large (N — 857) and this turns out to be a highly significant finding: Chi^2 — 43.3 and P ∠ .001, or the probability of this occurring purely by chance is less than 1 in 1,000.

There were 125 patients who had been previously self-discharged who were readmitted to the hospital in 1952. Approximately identical figures were found in the two samples of self-discharge and control population, but because of the larger number of patients of the entire sample of all admissions and readmissions, this was graphed.

The patient who goes out against advice returns sooner and his disease accordingly progresses more rapidly. Both the self-discharge and the control patients received the same intensity of medical treatment. Without regard to the specific types of therapy, the two samples were compared as to whether they received chemotherapy, air, surgery or any combination of these. The figures were almost identical. It should be pointed out, however, that the self-discharge patients who left before the first month of care was completed were excluded from the surgery tabulation.

TABLE V

Percentage of Patients Leaving According to Three-Month Periods of Hospitalization Before Self-Discharge and Type of Self-Discharge (Entire Hospital)

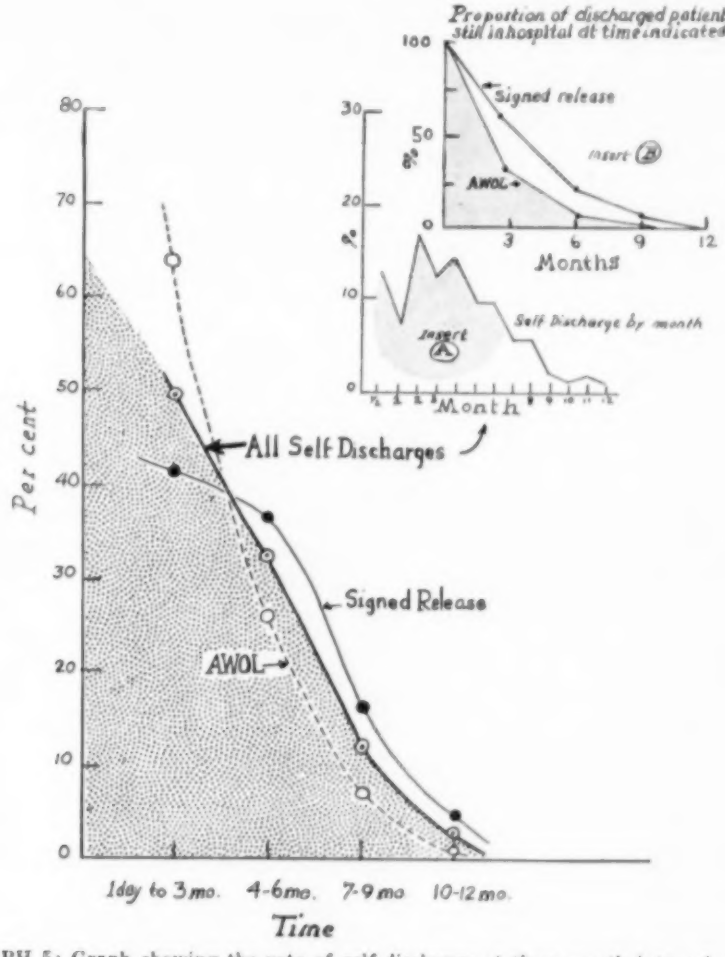
Span in Months	Signed Release		AWOL		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
1 to 3 months	47	41.2	42	64.6	89	49.7
4 to 6 months	42	36.8	17	26.2	59	32.9
7 to 9 months	19	16.7	5	7.7	24	13.5
10 to 12 months	6	5.3	1	1.5	7	3.9
Total	114	100.0	65	100.0	179	100.0

Percentage of Patients Leaving According to Months of Hospitalization Before Self-Discharge and Type of Self-Discharge (Entire Hospital)

Number of Months	Signed Release		AWOL		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Under 1 month	13	11.4	10	15.4	23	12.8
1	9	7.9	5	7.7	14	7.8
2	16	14.0	15	23.1	31	17.3
3	9	7.9	12	18.5	21	11.7
4	17	14.9	8	12.3	25	13.9
5	14	12.3	3	4.6	17	9.5
6	11	9.6	6	9.2	17	9.5
7	6	5.2	4	6.2	10	5.6
8	10	8.8	0	0.0	10	5.6
9	3	2.6	1	1.5	4	2.2
10	2	1.8	0	0.0	2	1.2
11	2	1.8	1	1.5	3	1.7
12	2	1.8	0	0.0	2	1.2
Total	114	100.0	65	100.0	179	100.0

When Do Patients Leave?

The greatest risk of self-discharge occurs within the first two months of hospitalization. Two-thirds of all the AWOLs were gone in three months, and 40 per cent of the Signed Releases will have left during the same period; that is to say, approximately 40 per cent of the problem lies in



GRAPH 5: Graph showing the rate of self-discharge at three-month intervals following hospitalization. AWOL patients leave more quickly, so the dotted line shows a more precipitous fall. Signed Release patients do not leave as quickly, so an S-shaped curve reveals the problem to exist over a six-month period. It is evident from this graph that the major self-discharge problem has already occurred at 4½ months, where the two lines cross each other. *Insert A* shows the percentage of all self-discharge patients leaving each month rather than by three-month periods; it shows a peak at two weeks and two months, with a decline in the first month. *Insert B* shows the proportion of self-discharged patients still in the hospital at the time indicated by three-month period. Thus, at three months the top line shows that about 60 per cent of the total Signed Release population still remains in the hospital, but only about 30 per cent of the AWOLs still remain.

the first two months of hospital stay (Table V). When this is graphed (Graph 5) one sees a very interesting phenomenon take place between admission and the first six months of hospital stay. The dotted line indicating the percentage of AWOL patients who leave in each three month period shows a precipitous fall in the first six months. The solid thin line showing Signed Release, on the other hand, shows an S-shaped curve, and while the majority of patients leave in the first three months the problem remains one of almost comparable magnitude for the first six months of stay. The lines cross at approximately four and one-half months, so that if one looks at the graph for this early period of hospitalization one is struck by the rapid decline in the AWOL group. If one disregards this early period the lines follow the same relative relationship.

STATISTICS TABLE NO. 4

At median (3 months)	Below	Above	Total
Signed Release	47	67	114
AWOL	42	23	65
Total	89	90	179

TABLE VI

Length of Hospital Stay and Classification of Pulmonary Tuberculosis

Category & Length of Stay	Minimal No.	Minimal Per Cent	Mod. Advanced No.	Mod. Advanced Per Cent	Far Advanced No.	Far Advanced Per Cent
Control sample:						
1-3 mos.	3	30.0	9	31.0	5	16.7
4-6 mos.	5	50.0	5	17.2	4	13.3
7-9 mos.	2	20.0	5	17.2	4	13.3
10-12 mos.	0	0.0	10	34.6	17	56.7
Total	10	100.0	29	100.0	30	100.0
All Self-Discharge:						
1-3 mos.	1	33.0	7	27.4	14	31.2
4-6 mos.	2	67.0	13	50.0	10	22.2
7-9 mos.	0	0.0	4	15.0	10	22.2
10-12 mos.	0	0.0	2	7.6	11	24.4
Total	3	100.0	26	100.0	45	100.0
Signed Release:						
1-3 mos.	1	50.0	6	35.3	10	30.3
4-6 mos.	1	50.0	8	47.1	8	24.2
7-9 mos.	0	0.0	1	5.9	7	21.3
10-12 mos.	0	0.0	2	11.7	8	24.2
Total	2	100.0	17	100.0	33	100.0
AWOL:						
1-3 mos.	0	0.0	1	11.1	4	33.3
4-6 mos.	1	100.0	5	55.6	2	16.7
7-9 mos.	0	0.0	3	33.3	3	25.0
10-12 mos.	0	0.0	0	0.0	3	25.0
Total	1	100.0	9	100.0	12	100.0

If one disregards the status of disease and considers only length of stay in the control and self-discharge groups, it is found that the self-discharged remain for a statistically significant shorter period of time. With a median self-discharge at three months the Signed Release and AWOL are compared according to the number staying less than the median and more than the median. In this instance it is found that the majority of the Signed Release stay more than three months and the majority of the AWOL stay less than three months. This difference is significant, yielding a Chi^2 of 9.06, $P < .01$.

Breaking down all self-discharges according to individual months of hospitalization, in Insert A of Graph 5 one sees a peak at two weeks and at two months. It is at two months and at three months that the AWOLs exceed the Signed Releases in the greatest proportion.

In Insert B one can see the percentage of self-discharge patients still in the hospital at each three-month period.

Although the number of cases is small in the categories in Table VI, showing the length of hospital stay and extent of tuberculosis, it gives a rough indication of the more advanced cases in the self-discharge group who leave the soonest, as compared to the controls or to all other self-discharges leaving in the later periods; that is to say, the problem in the early months of stay is not only the greatest numerically, but the sicker people leave the quicker. This is true in the far advanced cases. As to the moderately advanced ones, the peak comes in the second three-month period in each category.

One can conclude from these tables and graphs that the time to attack the problem of the self-discharge patient is in the very earliest weeks and months of hospitalization. Without a concerted attempt to meet this at the time when the danger is greatest, the horse will have fled before the barn door is closed.

Length of Hospital Stay

The self-discharge patients stayed an average of 4.4 months; the Signed Release portion stayed 5 months and the AWOL 3.5 months, on the average. The regular discharge patients remained in the hospital for an average of 8.7 months (Table VII Graph 6).

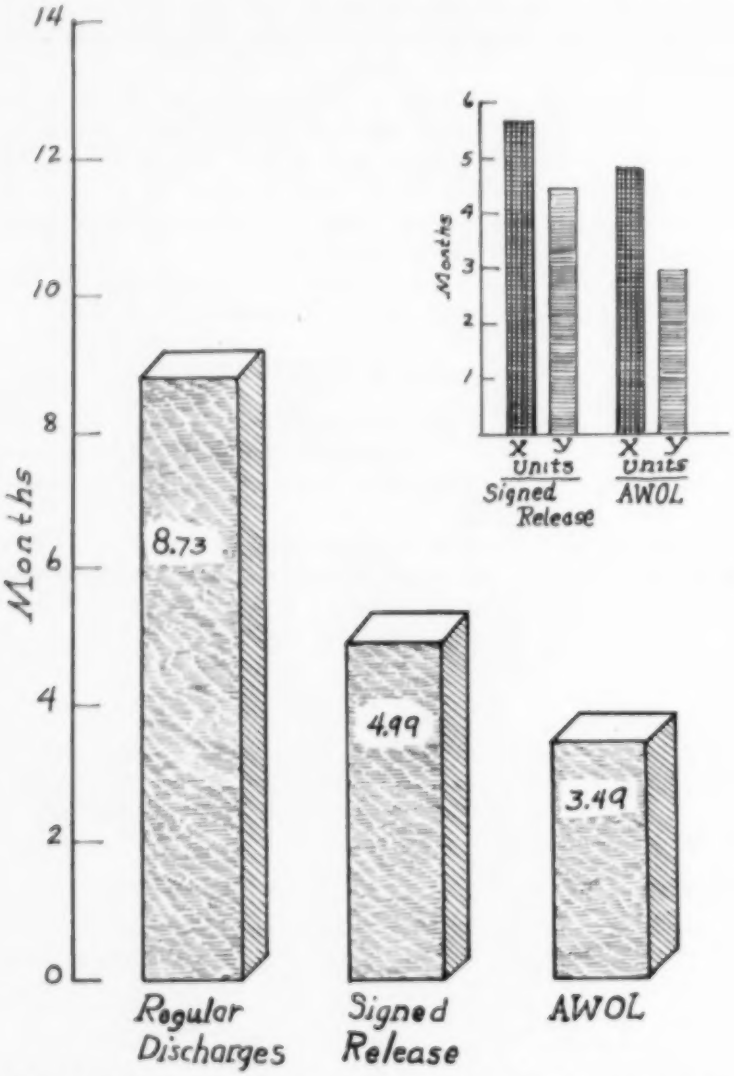
TABLE VII
Length of Hospital Stay Before Discharge (Entire Hospital)

Type of Discharge	Number of Patients	Total Months	Average Stay (mos.)
Regular Discharge (Control)	69	611*	8.73
Signed Release	118	589**	4.99 (5)
AWOL	67	234***	3.49 (3½)
Total Self-Discharge	184	823	4.44

*Range 1 to 36 mos.
**Range 1 to 24 mos.
***Range 1 to 18 mos.

Calendar Month of Self-Discharge

When the self-discharge patients were graphed according to calendar month of the year (Table VIII Graph 7) there was a decline in the number of self-discharge patients in the summer months. This was an unexpected finding, although the maximum variability between the lowest month of



GRAPH 6: Bar-diagram showing the number of months on the average that regular-discharge and self-discharge patients remain in the hospital. The insert shows the difference in duration of hospitalization for Signed Release and AWOL in the two units, showing that in Unit Y both Signed Releases and AWOLs stay for a shorter period.

self-discharge (July) and the highest (November) is only 8.1 per cent. Both the total admissions and the total live discharges show a much more even distribution without the summer drop (Table VIII).

Comparing the quarters of the year divided arbitrarily into seasons, one would assume that if there was no difference, each quarter would yield 25 per cent of the self-discharges. The summer months, however, show 17.9 per cent self-discharge. This difference is significant, giving a critical ratio of 2.23, $P < .03$. Both AWOL and Signed Release show the same decrease in self-discharge during the summer months.

TABLE VIII

Admissions and Discharges by Calendar Month (Entire Hospital)

Month	Total Admissions		Total Live Discharges		Total Self-Discharges	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
January	86	9.7	73	9.9	21	11.3
February	76	8.6	49	6.6	17	9.2
March	83	9.4	62	8.4	20	10.8
April	73	8.3	57	7.7	13	7.0
May	70	7.9	55	7.5	10	5.4
June	77	8.7	65	8.8	10	5.4
July	90	10.2	68	9.2	9	4.9
August	59	6.7	66	9.0	14	7.6
September	66	7.5	48	6.5	20	10.8
October	66	7.5	64	8.7	14	7.6
November	65	7.4	80	10.9	24	13.0
December	72	8.1	50	6.8	13	7.0
Total	883	100.0	737	100.0	185*	100.0
			By Season			
Winter	234	26.4	172	23.3	51	27.5
Spring	226	25.6	174	23.6	43	23.2
Summer	226	25.6	199	27.0	33	17.9
Fall	197	22.4	192	26.1	58	31.4
Total	883	100.0	737	100.0	185*	100.0

*The discrepancy between 184 and 185 for total self-discharge patients could not be rectified inasmuch as the latter figure was obtained in the month-to-month tabulation.

TABLE IX

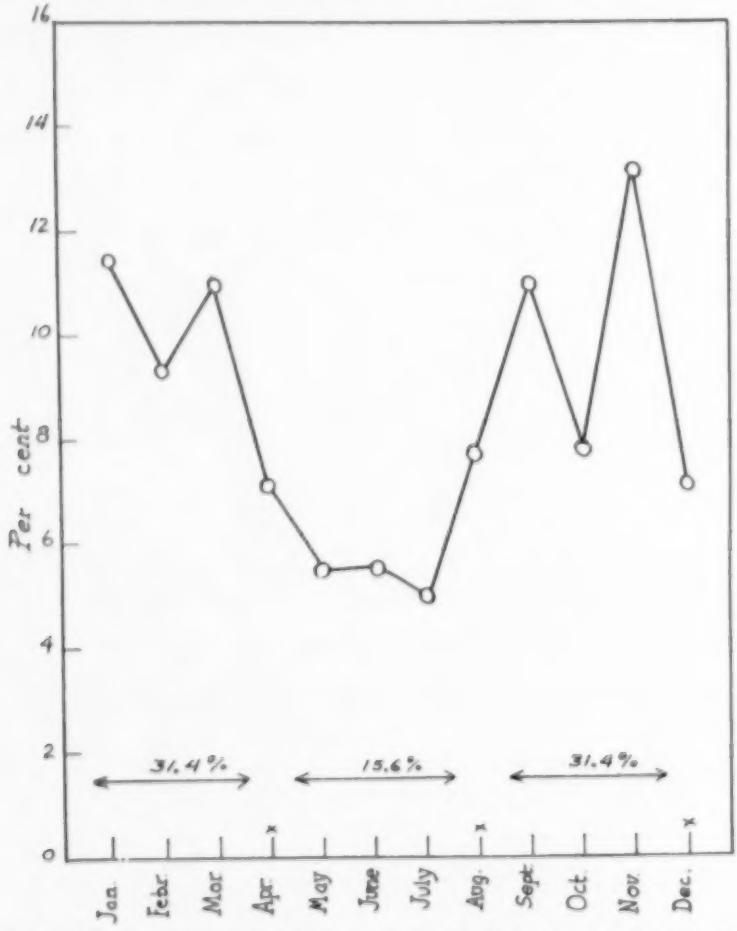
Self-Discharge by Favorable and Unfavorable Months Regardless of Season

Quartile	Average of Each Month Per Cent	Number	Average of Period Per Cent
Favorable months:			
1. May, June, July	5.2	29	15.6
Unfavorable months:			
1. January, February, March	10.4	58	31.4
2. September, October, November	10.4	58	31.4
Transition months:			
1. April, August, December	7.0	40	21.6
Total		185*	100.0

*The discrepancy between 184 and 185 for total self-discharge patients could not be rectified inasmuch as the latter figure was obtained in the month-to-month tabulation.

When one arbitrarily divides the seasons of the year (Table VIII) one has a statistical expression of this phenomenon, but more light is thrown on the problem by dividing the year into favorable and unfavorable months (Table IX). In the favorable months of May, June and July, 15.6 per cent of the self-discharge patients left the hospital. In the unfavorable months of January, February, March and/or September, October, November, 31.4 per cent left as self-discharged. In the three months of transition the percentage of self-discharge patients was approximately the same, or 7 per cent.

What does this mean? One might be inclined to speculate that the summer months would lead to more frequent self-discharge, when the



GRAPH 7: A line graph showing the percentage of self-discharge patients leaving the hospital at each calendar month. It is shown that a smaller percentage of patients leave against advice in the summer months. The favorable three-month period and the unfavorable three-month periods are indicated at the bottom, with an X before the transition months.

TABLE X
Marital Status of Control Sample and Self-Discharged

Category	No.	Per Cent
Controls:		
Single	15	21.7
Married	36	52.2
Separated or divorced	16	23.2
Widowed	2	2.9
Total	69	100.0
All self-discharged:		
Single	13	17.6
Married	17	23.0
Separated or divorced	38	51.3
Widowed	6	8.1
Total	74	100.0
Signed Release:		
Single	8	15.4
Married	12	23.1
Separated or divorced	27	52.0
Widowed	5	9.5
Total	52	100.0
AWOL:		
Single	5	22.7
Married	5	22.7
Separated or divorced	11	50.0
Widowed	1	4.6
Total	22	100.0

weather is at its best. This was not the case. On the other hand, one might think that the high fall self-discharge rate was a reflection of patients leaving for the holidays; but one can hardly then account for the fact that December is a low transition month, since this month would catch the Christmas and New Year absentees. Is this something related specifically to this hospital, this setting, this patient supply or what? One cannot answer on the data we have.

Marital Status

The marital status of the control and self-discharge patients was compared, and the striking observation made that the largest segment of patients in the controls was married and the largest segment among the self-discharged was separated or divorced (Table X Graph 8). Considering these two groups, they approximately reverse each other in the two patient samples. In the controls, for example, 23.2 per cent were separated

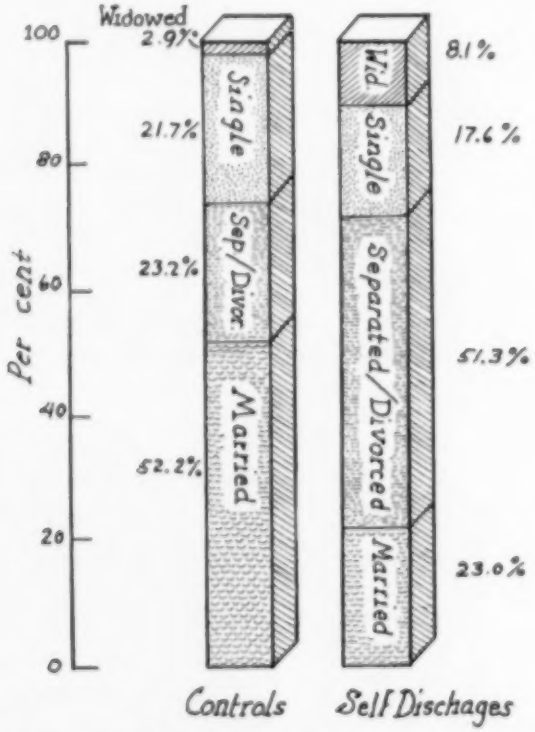
STATISTICS TABLE NO. 5

	Divorced/Separated	Married	Total
Control	16	36	52
Self-Discharge	38	17	55
Total	54	53	107

or divorced and 52.2 per cent were married, while in the self-discharged 23 per cent were married and 51.3 per cent separated or divorced. The majority of the controls were married, the majority of the self-discharged were separated or divorced. This is a highly significant finding: $Chi^2 = 15.7$, $P < .001$.

An enduring marriage seems to be a deterrent to self-discharge. Does this mean that in the control group those patients whose marriages are not broken are a more stable sample in regard to the factors that lead to other sorts of separation such as hospital confinement, while the converse is true for the self-discharged? Does it mean that the married patients remain in the hospital because of problems and responsibilities relating to the marriage, that is, spouse and children? If one considers these two groups as patients who have been married and therefore might have children, and disregards the number of children, it is found that 62 per cent of both groups have children; that is to say, the presence or absence of offspring is identical in the two groups.

The self-discharged patient married or previously married had an average of 2.39 children, while the controls had an average of 1.22 children



GRAPH 8: Bar-diagram showing the relative proportion of married separated/divorced, single and widowed patients in the control and self-discharge samples. This shows the majority of the self-discharged are separated or divorced, and the majority of the control are married.

(Table XI). It would be interesting to speculate on the greater number of children among the self-discharged patients; however, when tested statistically the difference did not prove significant. It would seem that while an enduring marriage is more likely to characterize the faithful patient and a broken marriage the self-discharge patient, the number of children is not important.

Siblings

The average number of siblings was approximately the same (2.5) for control and self-discharge patients.

Alcoholism

The case records were divided into two groups: those designated as alcoholic by the staff and those without a history of alcoholism. It is exceedingly difficult from the study of such records to say by what criteria the diagnosis of alcoholism was arrived at; some case records left no doubt. Since we are comparing two samples of patients diagnosed by the same staff, it is apparent that the same reasons for diagnosing alcoholism operated for the two groups. A few cases were removed from the alcoholic category because the designation of alcoholism appeared in passing comments noted on the chart or was made at the time of discharge after one episode of drinking.

The problem of alcoholism is a major one in the city of San Francisco. Drinking in the hospital is generally considered a serious infraction of sanatorium discipline. The problem is usually approached by hospital administrators in a punitive or moralistic manner. While the medical pro-

TABLE XI

Ratio of Children to Each Married, Divorced/Separated or Widowed Patient

Category	Ratio	No.
Controls:		59
Married	0.7 :1	
Separated or divorced	2 :1	
Widowed	0 :2	
Total (average number)	1.22:1	
All self-discharged:		63
Married	3.4 :1	
Separated or divorced	0.5 :1	
Widowed	1.5 :1	
Total (average number)	2.39:1	
Signed Release		44
Married	3.6:1	
Separated or divorced	0.4:1	
Widowed	2 :1	
Total	1.5:1	
AWOL:		19
Married	3 :1	
Separated or divorced	0.8:1	
Widowed	0 :1	
Total	1.3:1	

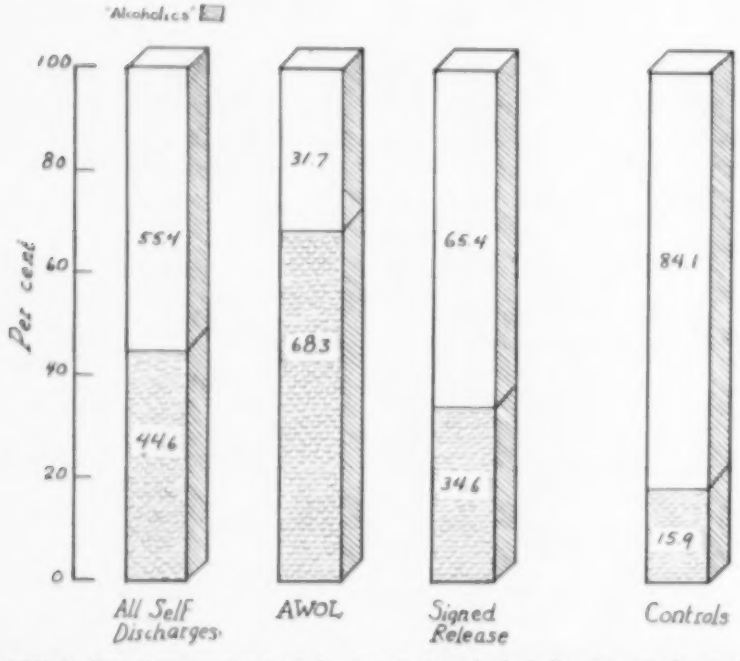
fession has come more and more to regard alcoholism as a disease, an illness, and not as a crime, the relationship of alcoholism and the way in which it is handled in a tuberculosis hospital is of great practical importance. In several other of the larger sanatoria there are Alcoholics Anonymous units in operation. This seems proper. In others the patients are locked out (Disciplinary Discharge) or locked in (in sanatorium prisons or in city or county jails).

The patient who has tuberculosis and who also suffers from alcoholism or periodic drinking is doubly burdened. If one separates from the group of alcoholics that portion whose alcoholism is of such magnitude, duration and history as to be relatively hopeless with our present means of therapy, there remains a group toward whom preventive and therapeutic techniques should be directed.

TABLE XII

Alcoholic and Non-Alcoholic Patient Sample According to Type of Self-Discharge

(Both Sexes)	Controls		All Self-Discharge		Signed Release		AWOL & Disc. Dis.	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Alcoholic	11	15.9	33	44.6	18	34.6	15	68.3
Non-alcoholic	58	84.1	41	55.4	34	65.4	7	31.7
Total	69	100.0	74	100.0	52	100.0	22	100.0



GRAPH 9: Bar-diagram showing the proportion of alcoholics among the control and self-discharge. This reveals the greater proportion of alcoholics (according to the definition in the text) among the self-discharged predominantly in the AWOL.

STATISTICS TABLE NO. 6

	Alcoholic	Non-Alcoholic	Total
Control	11	58	69
Self-Discharge	33	41	74
Total	44	99	143

There was a history of alcoholism in 44.6 per cent of all self-discharge patients (Table XII Graph 9) compared to 15.9 per cent in the control. The greater proportion of alcoholics in the self-discharged is significant. Patients with a history of alcoholism are three times as frequent in the self-discharged as compared to the control. The non-alcoholics are one and one-half times more numerous in the control than in the self-discharged: $Chi^2 = 13.8$, $P < .001$.

Thus, the problem of the alcoholic patient was of more than twice the magnitude in the self-discharged as compared to the control. If one considers the two subgroups it is to be noted that an alcoholic history was twice as great a problem in the patients going AWOL as in those who signed themselves out. Among the Signed Releases the non-alcoholics predominate 2 to 1, while among the AWOLs the alcoholics predominate 2 to 1. This is a significant difference: $Chi^2 = 7.05$, $P < .01$.

STATISTICS TABLE NO. 7

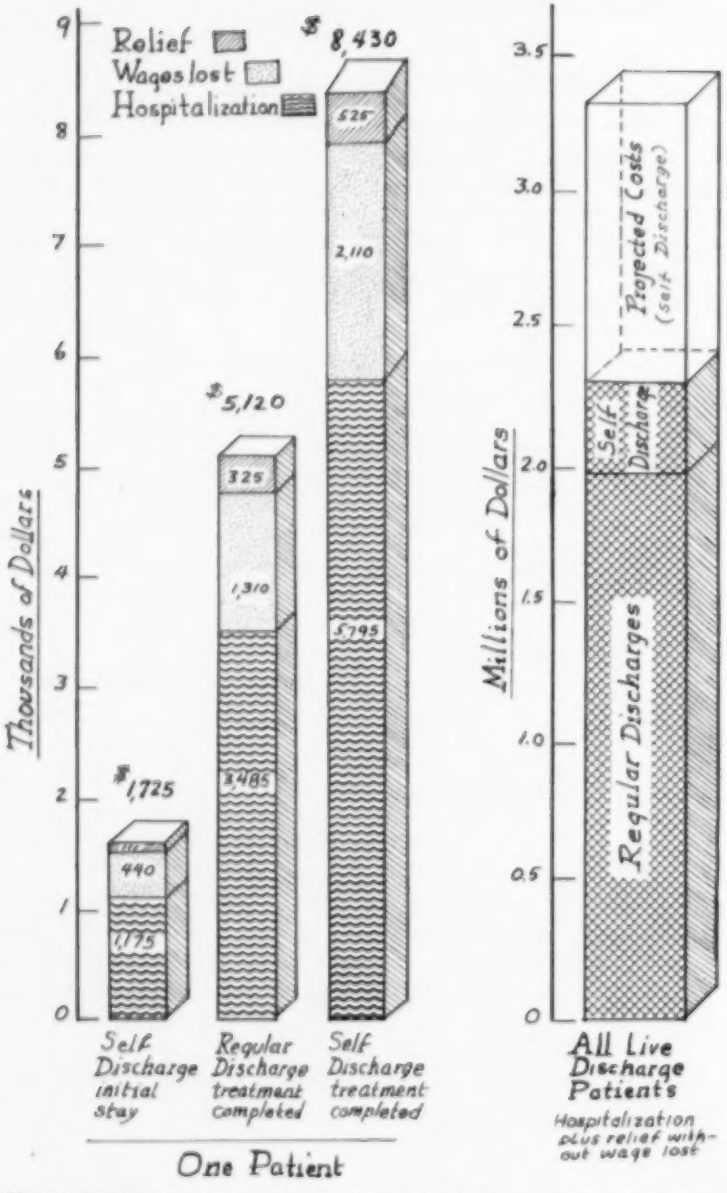
	Alcoholic	Non-Alcoholic	Total
Signed Release	18	34	52
AWOL	15	7	22
Total	33	41	74

Economics

To evaluate the cost to the City and County it is necessary to make some speculations as to future behavior of patients, and also some arbitrary stipulations. To make the following calculations the behavior of the 184 self-discharge patients was considered. On the basis of the per diem cost to the City of $8.82 per patient, multiplied by the average length of stay of the self-discharge sample, the cost for hospitalization amounted to $217,375 (Table XIII). On the basis of the average hospital stay of

TABLE XIII
Costs of Hospitalization, Lost Wages, Aid and Relief
For Entire Tuberculosis Hospital

Discharge	No. Pats.	Hospitalization	Aid	Total Excl. Wages	Wages Lost	Total
Regular Discharge (Treat. complete)	524	$1,826,140	$172,000	$1,998,140	$686,000	$2,684,140
Self-Discharge (Initial stay)	184	$ 217,375	$ 20,400	$ 237,775	$ 81,000	$ 318,775
Self-Discharge (Treat. complete)	184	$1,066,280	$ 97,000	$1,163,280	$389,000	$1,552,280



GRAPH 10: Graph showing the cost in thousands of dollars to the City, County and State for hospitalization of self-discharge and regular-discharge patients, and in millions of dollars for all live discharged patients for 1952: 524 regular-discharge and 184 self-discharge; with an estimated projected cost for self-discharge patients in excess of a million dollars, a considerable portion of which could be saved.

TABLE XIV
Cost Per Patient for Hospitalization, Lost Wages, Aid and Relief

Discharge	Hospitalization	Aid	Total Excl. Wages	Wages Lost	Total
Regular Discharge (Treatment completed)	$3,485	$325	$3,810	$1,310	$5,120
Self-Discharge (Initial stay)	$1,175	$111	$1,286	$ 440	$1,730
Self-Discharge (Treatment completed)	$5,795	$525	$6,320	$2,110	$8,430

the regular discharge patients (corrected for their hospital stay after transfer to other institutions) for a total of 13.1 months, the cost for 524 regular discharge patients was $1,826,140. The cost of hospitalization per self-discharge patient was $1,175 compared to $3,485 per regular discharge patient (Table XIV). Disregarding the fact that a certain number of routine discharge patients *will* return to the hospital for subsequent care, attention is focused on the money spent for the total care of the self-discharge sample of 184 patients. It is with these patients, who interrupt their treatment and represent loss in money, time and effort that savings can be considered. Assuming from our sample study that the self-discharge patient on the average will have two self-discharge and one regular discharge hospitalizations, the figure for the care of the 184 patients becomes $1,066,280, or $5,795 per patient. This represents an added burden in the vicinity of $2,000 per patient, or a total of $368,000 per year, in comparison to a similar number of routine discharge patients. This figure will be diminished somewhat by the cost of rehospitalization of some routine discharge patients; if it were reduced to one-quarter of a million dollars it represents a potential economy of major proportions.

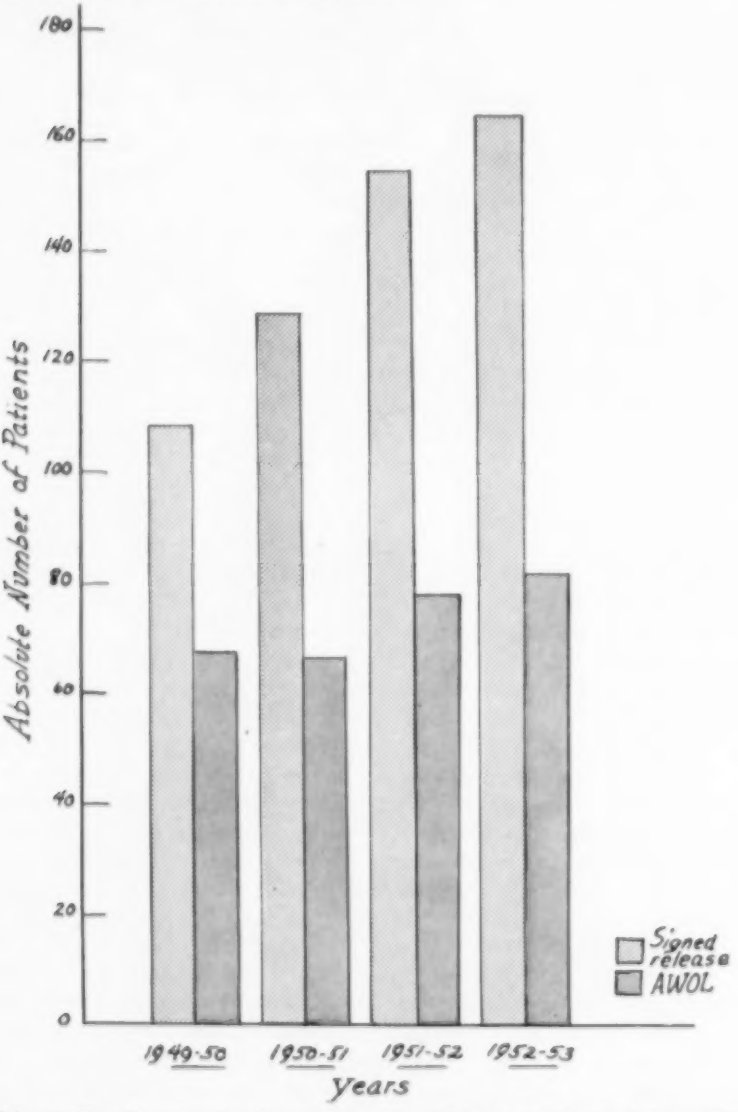
Often hospitals are thought of by budgeting bodies somewhat as hotels are: as long as the beds are kept full they are functioning adequately and doing a service, but over a five year period many of these tuberculosis patients will be repeatedly occupying a hospital bed when they might otherwise be productive citizens. By a conservative estimate the cost of the self-discharge patient over a five year period represents an excess liability to the hospital of over one and one-fourth million dollars (Graph 10).

The cost in suffering and social welfare is no less striking. The question of whether or not the budget of a hospital of such size can support an additional social worker becomes meaningless. If one adds to the cost of hospitalization the items of public financial aid and wages lost, the total cost for the regular discharge patient is $5,120 and for the self-discharge $8,430 (Table XIV). Here is an added burden of $3,000 per patient. These figures were arrived at by the observation that in the self-discharge sample studied, 50 per cent were previously employed for an average income of $200 per month, and public aid from all sources was $100 per patient per month for 25 per cent of the self-discharged patient population.

If the number of self-discharge patients were reduced by one-quarter,

the savings in the initial stay would amount to $54,343, or for their projected care, $266,570. While it is true that some patients will go home and not return for further hospital care, it is also true that some will return and remain for years or for the rest of their lives.

One of the most important needs is a full-time medical director for the tuberculosis section of the hospital.



GRAPH 11: Bar-diagram showing increase in self-discharge in absolute numbers in 4-year periods since 1949, particularly striking in Signed Release. When computed in terms of percentage of admissions and percentage of live discharges, the proportion of self-discharges is still steadily rising.

SUMMARY

The self-discharge patients from the San Francisco City and County Hospital were studied for the year 1952. There were 184 self-discharged patients and 524 regular-discharge patients from the entire Tuberculosis Hospital. A detailed study of all the self-discharges (74) and a control sample (69) from the Stanford service were compared statistically.

It was evident from the data that the patients who go out as Signed Release are different from those who go Absent Without Leave (AWOL) in observable ways.

An attempt has been made to define what characterizes the self-discharge sample from medical and social viewpoints. One can neither plan nor evaluate programs of patient care when one does not know the nature of the patient sample.

It is emphasized that the patient sample, as well as the administration, vary from hospital to hospital and from year to year. Errors have been committed in formulating generalizations about tuberculosis patients without cognizance of the difference between hospitals, patient samples and also without realizing that the self-discharge population consists of two important subgroups.

There were 30.7 per cent self-discharge patients, the majority of whom had far advanced disease. The majority of the control did not have far advanced disease.

There was no sex difference between the total self-discharge and the total control population.

The female self-discharges had a significantly greater incidence of positive sputum than the male self-discharges. The male AWOLs had a significantly greater positive sputum incidence than the female AWOLs.

There was a highly significant increase in the extent of disease among readmitted self-discharge patients compared to all first admissions.

The majority of self-discharge patients left within the first six months. The majority of AWOLs stayed less than three months! The majority of Signed Releases stayed more than three months. The self-discharged left sooner than the control.

There was a decrease in number of self-discharges during the summer months, which was statistically significant.

The majority of self-discharges were separated or divorced, while the majority of controls were married. This was highly significant statistically.

A history of alcoholism was three times greater in the self-discharged as compared to the control. In the AWOLs the alcoholics predominated, while in the Signed Releases the non-alcoholics predominated. These were significant differences.

It is estimated that the cost of hospitalization to the city, county and state for the 184 self-discharge patients of the entire Tuberculosis Hospital was $217,375. In their projected cost based on readmission figures it was estimated that the self-discharge sample over a five-year period represents a financial liability in excess of one and one-quarter million dollars.

It is estimated that their behavior as self-discharge compared to regular discharge patients represents an added burden of approximately $2,000 per patient.

If the self-discharge population were reduced by one-fourth, the savings in hospitalization would amount to $54,343 a year, or over one-quarter of a million dollars in five years. To spend money to ameliorate this situation would not only be humanely wise but economically sound.

If it would be possible to define the patient population at risk from the standpoint of self-discharge, then it would be possible to compare the changes in risk factors over a period of time and in relation to adjustment of program.

The lack of statistical control studies of such an important part of the tuberculosis program should spur students of the problem to extensive and careful studies.

RESUMEN

Se estudiaron las salidas voluntarias de los enfermos del hospital de la Ciudad y el Condado de San Francisco durante el año de 1952.

Hubo 184 altas voluntarias y 524 altas regulares de enfermos, en todo el hospital de tuberculosis. Se compararon estadísticamente todas las altas voluntarias (74) y una muestra de control (69) del servicio de Stanford.

Fué evidente de acuerdo con estos datos que los enfermos que salieron con alta voluntaria firmada, son diferentes de los que salen con permiso y no regresan.

Se hizo un intento para determinar qué características tiene el grupo de las altas voluntarias desde el punto de vista médico y social.

No se quede planear ni valorar un programa de cuidado de enfermos cuando no se conoce la naturaleza de la muestra de enfermos.

Se recalca que la muestra de enfermos así como la administración, varían de hospital a hospital y aún con año. Se han cometido errores al hacer-generalizaciones acerca de los enfermos de tuberculosis sin reconocimiento de las diferencias entre hospitales, muestras de enfermos y tambiénsin darse cuenta de que las altas voluntarias constan de dos subgrupos-importantes.

Hubo 30.7 por ciento de altas voluntarias, la mayoría de los cualestenían tuberculosis muy avanzada. La mayoría del grupo de control no tenía tuberculosis muy avanzada. Sin embargo, si se considera la clasificación en mínima, moderadamente avanzada y muy avanzada, más bien que la-simple división en muy avanzados y no muy avanzados, no hay diferencia-significante entre el control y las altas voluntarias.

No hubo diferencia de sexo entre el número total de altas voluntarias y el total control de la población.

El grupo de mujeres que salieron con alta voluntaria, tuvo una mayorincidencia significativa con esputos positivos qque el grupo de hombres.

Los hombres que salieron con permiso y no regresaron, tuvieron una incidencia de esputos positivos mayor que las mujeres en el grupo análogo.

Hubo un aumento de significación en la extensión de la enfermedad entre los readmitidos desqués de alta voluntaria que entre los admitidos por primera vez.

La mayoría de los enfermos que salieron voluntariamente, lo hicieron dentro de los primeros seis meses. La mayoría de los que salieron con permiso y no volvieron, habían estados menos de tres meses; la mayoria de los que se dieron de alta a petición firmada, estuvieron más de tres meses. Las altas voluntarias dejaron el establecimiento más pronto que los del grupo de control.

Hubo un decrecimiento del número de altas voluntarias durante el verano, que fué estadísticamente de significación.

La mayoría de las altas voluntarias correspondían a personas divorciadas o separadas, en tanto que la mayoría de los controles, eran casados.

Esto fué altamante significante desde el punto de vista estadístico.

Los antecedentes de alcoholismo, fueron tres veces más frecuentes entre los salidos voluntariamente que en los de control. Entre los que no volvían con después de permiso, dominaban los alcohólicos en tanto que entre los que salieron a solicitud firmada dominaban los no alcohólicos. Estas diferencias fueron de significación.

Se estima que el costo de hospitalización para la Ciudad, el Condado y el Estado para los 184 enfermos que se dieron de alta voluntariamente, fué de Dlls. 217, 375. Su costo se proyecta sobre la readmisión de modo que el grupo de muestra de estos enfermos que se dan de alta voluntaria, representa una pérdida de más de un cuarto de millón de dólares en cinco años.

Se estima que su conducta como enfermos que se dan de alta voluntaria en comparación con los enfermos que salen de manera regular, representa un costo adicional por enfermo aproximadamente de Dlls. 2.000.

Si el número de altas voluntarias se redujese a la cuarta parte las economías en los gastos del hospital serían de 54.343 Dlls. al año, o más de un cuarto de millón en los cinco años. Gastar dinero para mejorar esta situación, sería no sólo humanitario, sino económicamente justificado.

Si fuese posible determinar qué parte de los enfermos están en riesgo desde el punto de vista de las altas voluntarias, sería posible comparar lost cambios en los factores de riesgo sobre cierto tiempo en relación con el apegamiento al plan.

La falta de estudios de control estadístico en tan importante parte del plan contra la tuberculosis, debe animar a los estudiosos del problema para hacer más aplias y cuidadosas investigaciones.

RESUME

L'auteur a étudié les malades qui ont quitté de leur plein gré l'Hôpital de la Ville et du District de San Francisco au cours de l'année 1952. Dans tout l'Hôpital tuberculeux, il y eut 184 malades qui le quittèrent spontanément, ontre 525 qui sortirent munis de l'autorisation régulière. Une étude détaillée de tous les malades partis irrégulièrement (74) et un groupe de malades, pris à titre d'exemple dans le service Stanford, furent comparés au point de vue statistique.

Les constatations faites démontrèrent que les malades qui quittent l'Hôpital par autorisation régulière diffèrent à plusieurs titres de ceux qui partent irrégulièrement. L'auteur a essayé de définir les caractéristiques des malades sortant irrégulièrement, des points de vue médicaux et sociaux. On ne peut en effet établir ni statistique ni conduite thérapeutique quand on ne connait pas la catégorie du malade. L'auteur insiste sur le fait que la catégorie du malade, tout comme l'administration elle-même, varie selon l'hôpital et selon l'année. On a commis des erreurs chaque fois que l'on a voulu généraliser au sujet des tuberculeux, sans tenir compte de la différence qui existe entre les établissements hospitaliers, la catégorie du malade, et le fait que les malades qui quittent l'hôpital sans autorisation se divisent en deux groupes importants.

Il y eut un pourcentage de 30.7% de malades qui quittèrent l'hôpital irrégulièrement. La majorité d'entre eux étaient porteurs de lésions très avancées. La majorité des malades régulièrement contrôlés n'avaient pas de lésions avancées.

Il n'y eut aucune différence selon le sexe des malades dans l'ensemble des malades indisciplinés ou régulièrement suivis.

Il y eut une plus grande fréquence d'expectorations positives chez les sujets féminins qui quittèrent l'hôpital irrégulièrement que chez les sujets masculins.

Les lésions sont généralement plus importantes chez les malades qui sont réadmis après avoir quitté l'Hôpital d'eux-mêmes que chez ceux qui sont admis á l'Hôpital pour la première fois.

La majorité des malades sortant irrégulièrement quittent l'Hôpital dans les premiers six mois. La majorité de ceux-ci restèrent moins de trois mois. La majorité des malades régulièrement sortis firent un séjour de plus de trois mois. Les malades sortant irrégulièrement quittèrent donc l'hôpital beaucoup plus tôt que les autres.

Il y eut une diminution du nombre des malades sortant irrégulièrement pendant les mois d'été, qui se montra statistiquement valable.

La majorité des malades sortant irrégulièrement étaient séparés ou divorcés, tandis que la majorité des malades disciplinés étaient mariés. Ceci eut également une réel le importance statistique.

L'alcoolisme fut noté trois fois plus souvent chez les malades sortis irrégulièrement que chez les malades disciplinés. Chez les malades sortis irrégulièrement, il y eut beaucoup plus d'alcooliques que chez les autres.

On a évalué que le coût de l'hospitalisation pour la cité, le district et l'état en ce que concerne les 184 malades sortis d'eux-mêmes était de 217.-315 dollars. Dans le projet financier, basé sur les chiffres de réadmissions, on a estimé que le cas du malade sorti irrégulièrement, représente une charge financière excédant un quart de million de dollars pour une période de 5 ans.

On évalue que le comportement des malades indisciplinés comparés aux sortants réguliers représente approximativement une charge supplémentaire de 2.000 dollars par malade.

Si l'ensemble des malades indisciplinés était réduit du quart, les économies d'hospitalisation s'élèveraient à 54.343 dollars par an, et à 250.000

dollars pour une période de cinq ans. Dépenser de l'argent pour améliorer cette situation serait non seulement sage due point de vue humain, mais sain du point de vue économique.

S'il devenait possible d'établir le pronostic d'un groupe de malades en partant de la notion essentielle de l'indiscipline, il y aurait alors la possibilité de comparer les modifications des facteurs de pronostic pendant une certaine période de temps, et en fonction des modifications du programme.

L'absence d'études et de contrôles statistiques sur une partie aussi importante due programme tuberculeux devrait inciter les chercheurs à étudier le problème d'une facon approfondie et soigneuse.

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Experiences in the Surgical Alleviation of Congenital Pulmonic Stenosis*

HENRY T. NICHOLS, M.D., and DANIEL F. DOWNING, M.D., F.C.C.P.
Philadelphia, Pennsylvania

Since the introduction of the first successful operative procedure by Blalock and Taussig[1] for the alleviation of the effects of the tetralogy of Fallot there have been numerous procedures devised by various workers in an attempt to bring about a lower operative mortality, better clinical improvement, and a more physiological circulation.

Our experience in this field involves 82 cases of tetralogy of Fallot, 18 of isolated pulmonic stenosis, and 15 of pulmonic stenosis with patent foramen ovale. The tetralogy group were divided as follows: 56 infundibular, 18 valvular, 8 combined infundibular and valvular. The total of 115 cases represents the patients coming under the direct care of the junior author at Hahnemann Hospital, Philadelphia, and who were all treated by the direct approach of Sellors[2] and Brock.[3, 4]

We have preferred the direct procedure as the treatment of choice in tetralogy except in those presenting a long, narrow right ventricular outflow tract instead of an obstructing infundibular septum, and cases with a markedly hypoplastic pulmonary artery. We feel that our operative mortality of 10.9 per cent in the 82 cases of tetralogy compares favorably to the reported mortality of the shunting procedures,[5, 6] and our clinical results have been satisfactory. The operative mortality for the total 115 cases was 9.7 per cent.

In the treatment of tetralogy by the direct approach, we routinely explore the right ventricular cavity by the passage of a probe into its lumen

FIGURE 1: Pituitary type rongeur with straight shaft.

*From the Departments of Thoracic Surgery and Pediatrics, The Hahnemann Medical College and Hospital, and The Bailey Thoracic Clinic, Philadelphia, Pennsylvania. Presented at the 20th Annual Meeting, American College of Chest Physicians, San Francisco, California, June 17-20, 1954.

to palpate an infundibular ridge and to determine its extent if present. The probe is also passed out into the pulmonary artery to determine the presence or absence of valvular stenosis.

We have previously used a pituitary type rongeur (Figure 1) to resect any obstructing infundibular ridge. During the use of this rongeur, we have recognized two distinct disadvantages. First, in some cases the ridge approached to the anterior wall of the right ventricle so closely that the rongeur had to be opened before the jaws were fully within the ventricular cavity. This resulted in excessive blood loss, especially when it was difficult to identify the ridge quickly to permit the opened jaws to be applied accurately and rapidly to its margin. Second, there seemed to be an inherent tendency for this straight-shafted rongeur used from the left side of the table to point toward the aortic valve and sinuses of Valsalva (Figure 2). Thus, there is risk of injury to the aortic cusps or aortic wall.

In an attempt to overcome these handicaps, we have designed a new rongeur (Figure 3) with a laterally curved shaft so that, when in place within the right ventricle, the end is pointed to the left away from the aorta. The jaws agulate upward from the shaft of the instrument instead

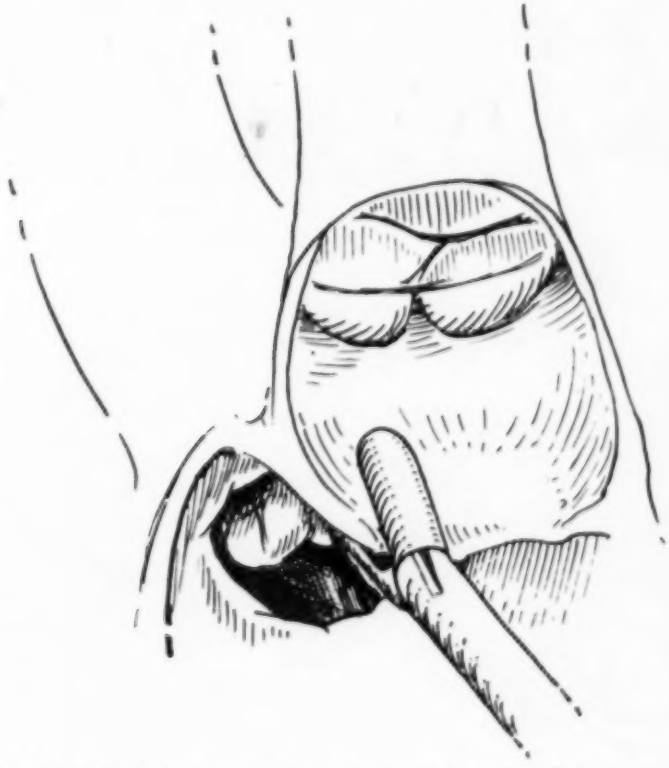

FIGURE 2: Straight-shafted rongeur has a tendency to point toward the aortic valve.

of being a straight extension of it, and it has an overhanging, stable, upper jaw. This permits the closed instrument to be passed into the infundibular chamber and then to be drawn back toward the apex so that the lower edge of the ridge can be identified as the shorter lower jaw falls over this edge and allows the upper jaw to rest on the top of the ridge. The rongeur then is opened and advanced to engage the lower, hawkbeak, jaw upon the ridge. The distal end of the lower jaw engages the ridge first and digs into the "embankment" in a steamshovel-like manner on closure (Figure 4).

This rongeur has been successfully used in the last few cases and we feel that it has given us a technical advantage over the use of the previously used pituitary rongeur.

In the treatment of pulmonary valvular stenosis, we have used both the

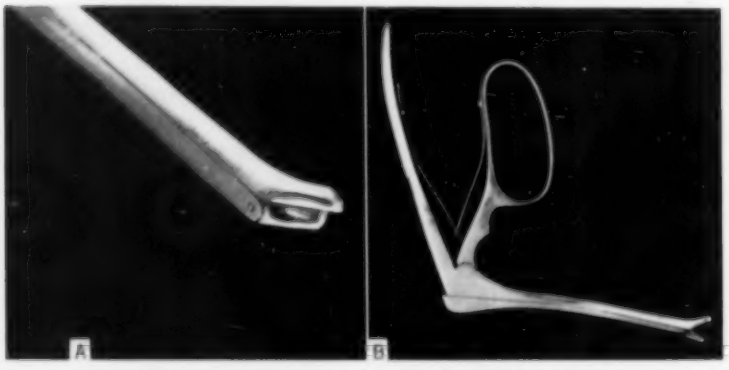

FIGURE 3A FIGURE 3B

Figure 3A: Jaws of new rongeur angulate from the shaft; lower jaw short and movable.—*Figure 3B:* Shaft of new rongeur sweeps to the right so that jaws point laterally, away from aortic valve.

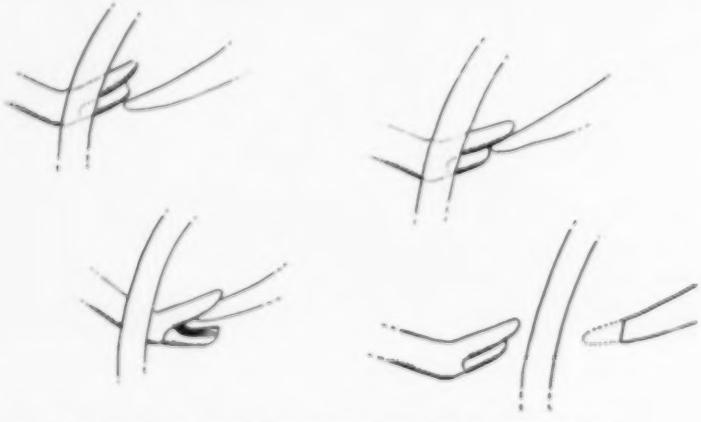

FIGURE 4: Infundibular resection with the new rongeur.

Potts valvulotome and a flat, diamond-shaped valvulotome with a malleable shaft.[7] Although the latter instrument (Figure 5) requires a larger ventricular incision, we have preferred it because of its malleable shaft which allows entrance through the right ventricular wall at a perpendicular angle and then a sweep in an arc to the pulmonary valve. Neither of these instruments has proved to be entirely satisfactory in our hands. All diamond-beaded valvulotomes seem to have a tendency to exert their greatest cutting force to the operator's right, to the lateral aspect of the pulmonary artery (Figure 6).

On three occasions in which there was heavy fibrosis of the valve which required increased pressure on the valvulotome, the tendency to cutting to the right has resulted in opening of the lateral wall of the pulmonary

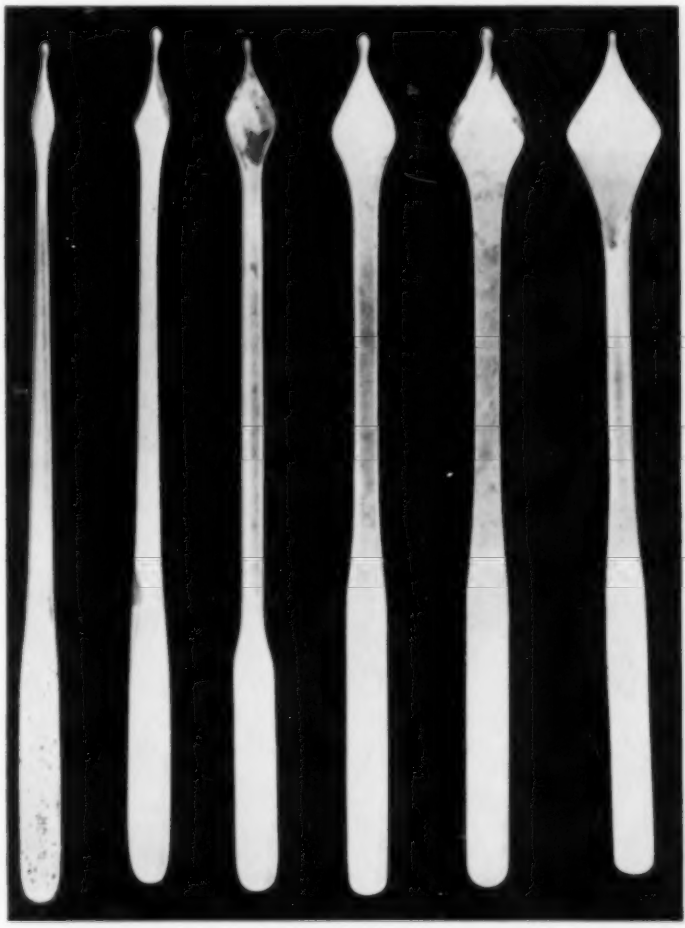

FIGURE 5: Flat, diamond-shaped valvulotomes with malleable handles.

artery. Although the incision in the artery wall was successfully sutured in each case, there was excessive blood loss.

Dr. Joseph Gale of Madison, Wisconsin, first brought this phenomenon to the attention of one of our group.[8]

This tendency toward unilateral cutting of the valve again was exemplified in a patient who came to autopsy several months following surgery. This patient had a pulmonary valvulotomy and later a right upper lobectomy for tuberculosis. Subsequent to this latter operation, the patient developed a bronchopleural fistula and died. At autopsy, it was found that unilateral cutting of the valve had occurred (Figure 7).

Such unilateral valvular cutting undoubtedly has allowed residual stenosis to remain in some cases. Lurie[9] and others have indicated that with this technique there remains a great, persistent pressure gradient between the right ventricle and the pulmonary artery on postoperative catheterization. We, too, have observed these postoperative findings. Some workers already have resorted to an "open technic" to insure complete valvular opening to the pulmonary ring under direct visualization. Dodrill[10] has utilized a bypassing pump to enable visualization of the pulmonary valve, and Swan et al[11] have utilized hypothermia plus temporary interruption of the circulation to make a visually guided attack upon the stenosed valve. Dodrill has pointed out the danger of his approach in a case diagnosed as isolated pulmonic stenosis in which there might actually have been an interventricular communication but with a right ventricular pressure equaling that of the left ventricle and no demonstrable shunt on cardiac catheterization. Death apparently resulted from cerebral air embolism.

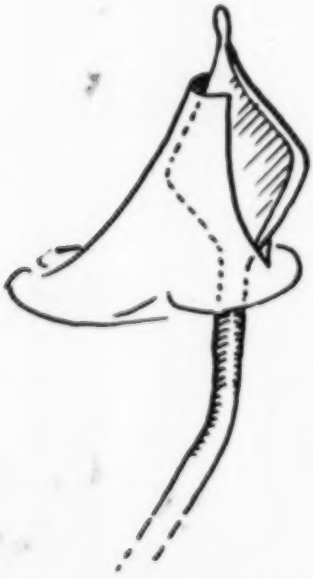

FIGURE 6: Diamond-shaped valvulotome has tendency to cut to the right.

It is of prime concern not to relieve the pulmonic obstruction completely in patients with the tetralogy of Fallot as this will result in flooding of the pulmonary vascular bed and possibly fatal pulmonary edema. For this reason alone, we feel that it is essential to explore the right ventricular cavity with a probe to be sure that no interventricular communication exists before proceeding with the valvulotomy. On two occasions, we have found an interventricular defect which had not been demonstrated by preoperative catheterization findings.

It was our feeling that, if the valve could be incised adequately all the way to the pulmonary arterial wall by a "closed technique" it would offer greater advantages of simplicity and safety than the "open technique" utilized by either Dodrill or Swan. Furthermore, it would seem to lessen the likelihood of producing pulmonary regurgitation which has been reported when a more extensive plastic procedure has been carried out upon the valve by the open technique.

Therkelsen[12] has devised a special clamp which permits secure attachment of a tube of latex to an opening made in the wall of the pulmonary artery. By the use of this tube, various cutting instruments may be introduced into the pulmonary arterial lumen for direct division of the stenotic valve in retrograde fashion.

After performing a valvulotomy on January 25, 1954 on R. S. (54-00612), a 21 year old white male at the Hahnemann Hospital with pure or isolated pulmonic stenosis via the Brock approach and utilizing a flat, diamond-shaped valvulotome, the ungloved left index finger then was inserted into the right ventricle to palpate the resultant valvular incision.

FIGURE 7: Pulmonary valve viewed from pulmonary arterial side, showing unilateral incision produced by diamond-shaped valvulotome.

Although a valvulotome estimated to be large enough to insure complete incision to the pulmonary ring on each side had been used, it was quite obvious that such a result had not been obtained. The greater part of the incision extended toward the left wall of the pulmonary artery, but valvular division was incomplete on both sides. The right and left Bailey mitral guillotine knives then were inserted successively along the left index finger to complete the incision to the artery wall bilaterally. It should be noted that Dr. Julian Johnson[13] independently, has performed the same procedure with the same mitral guillotines. Comparison of the pre- and postoperative catheterization tracings in this patient shows almost complete abolition of the pressure gradient between the right ventricle and pulmonary artery (Figure 8). The pressure in the right ventricle following surgery has fallen to within normal limits.

We felt that the guillotines could insure complete valvular incision but that they should be redesigned so that the valvulotomy could be carried out by a completely blind technique and without the help of the guiding finger. Certainly, the hearts of small children would not admit the surgeon's finger. Also, even if the blind approach were carried out in adults, it would insure a smaller ventricular incision. The only obstacle to the success of such a technique which really needed to be overcome seemed to be assurance that the guillotine knife could be engaged safely through the tightly stenosed opening.

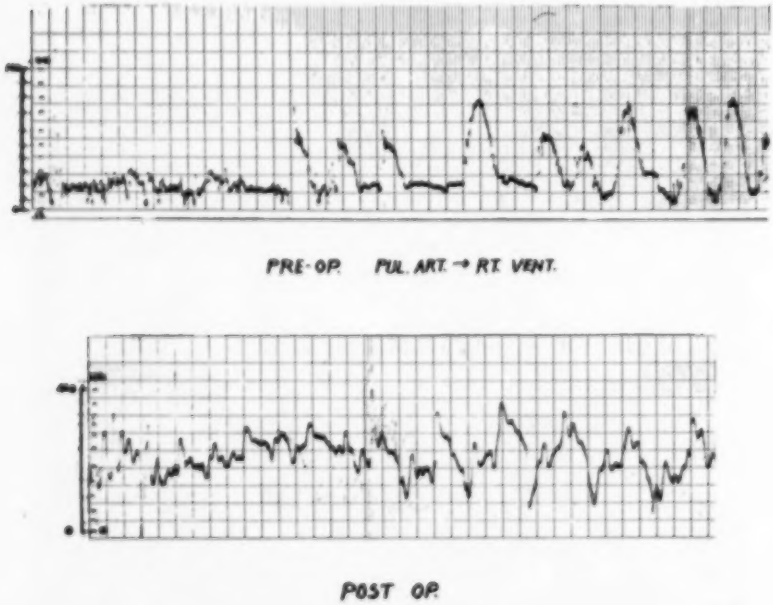

FIGURE 8: Pre- and postoperative continuous cardiac catheterization curves between pulmonary artery and right ventricle in Case 1. (Taken as catheter is withdrawn from the pulmonary artery into the right ventricle—right to left.)

The redesigned guillotine (Figure 9) has an olive tip to assure easy engagement within the valve opening. The oblique leading edge just proximal to the tip has a sharp edge so that it will partially incise the valve if such enlargement is needed to pass it through to the pulmonary artery. The guillotine has no other cutting edge when closed. After the instrument has been passed well above the valve, the guillotine is opened and as the instrument is pulled in a retrograde direction the tip is caused to deviate laterally toward the artery wall. The valve lip falls into the defect of the opened guillotine and the stable "beak" of the open knife engages the pulmonary artery side of the valve up to the pulmonic ring. The guillotine blade is then closed to incise the valve. It should be noted that the cutting edge of the movable blade is made angulated so that it first engages the valve peripherally at the pulmonic ring, thus insuring incision from this point to the central stenosed orifice (Figure 10). Then, the

FIGURE 9: Right and left guillotine knives designed for pulmonary valvulotomy.

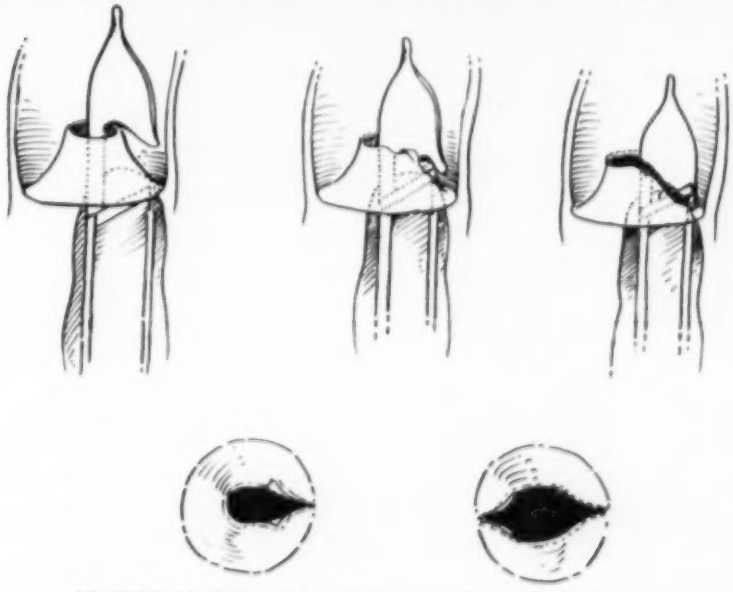

FIGURE 10: Pulmonary valvulotomy using guillotine knives.

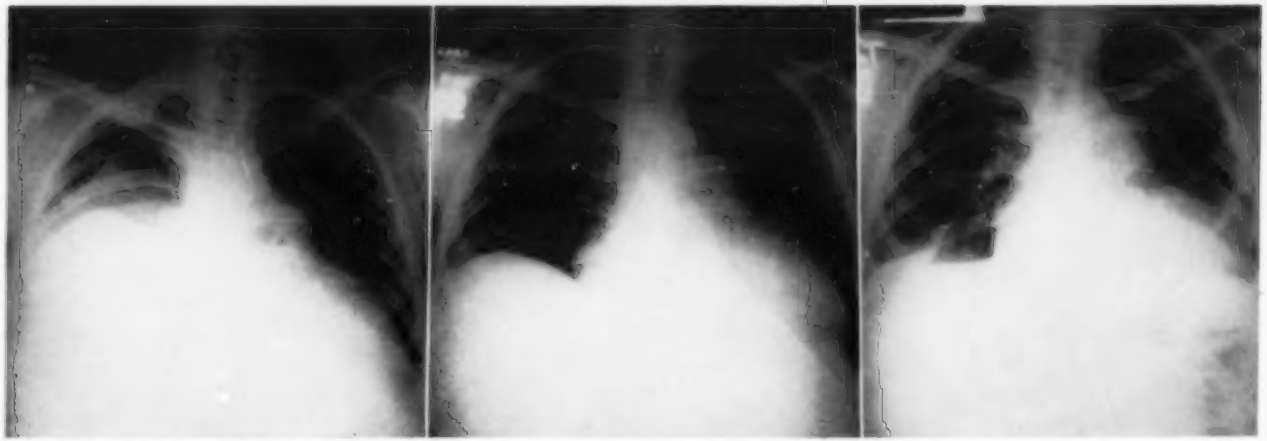

FIGURE 11A FIGURE 11B FIGURE 11C

Figure 11A, (Case 2): Admission x-ray.—*Figure 11B,* (Case 2): Immediate preoperative x-ray.—*Figure 11C,* (Case 2): Discharge x-ray.

closed instrument is removed. A second guillotine which cuts to the opposite side is inserted to complete the bilateral incision of the valve.

The second case, also one with isolated pulmonic valvular stenosis was operated by the guillotine knife approach without the aid of the guiding finger and using the knife described above. He was a 31 year old white male who was admitted in marked congestive failure and with gallop rhythm. His initial x-ray film (Figure 11A) revealed cardiomegaly and right pleural effusion, plus partial right pneumothorax which had been caused by thoracentesis performed prior to hospitalization. Rigid medical therapy and thoracenteses, reduced the overt evidence of congestive failure but it never disappeared. Gallop rhythm persisted throughout his six weeks preoperative stay. Pulmonary valvulotomy was carried out and no digital examination of the valve was done as he was considered a poor operative risk and it was felt that the least operative trauma and shortest anesthesia were imperative for his survival.

Postoperatively, evidence of congestive failure continued. Persistent reaccumulation of pleural fluid required numerous thoracenteses. How-

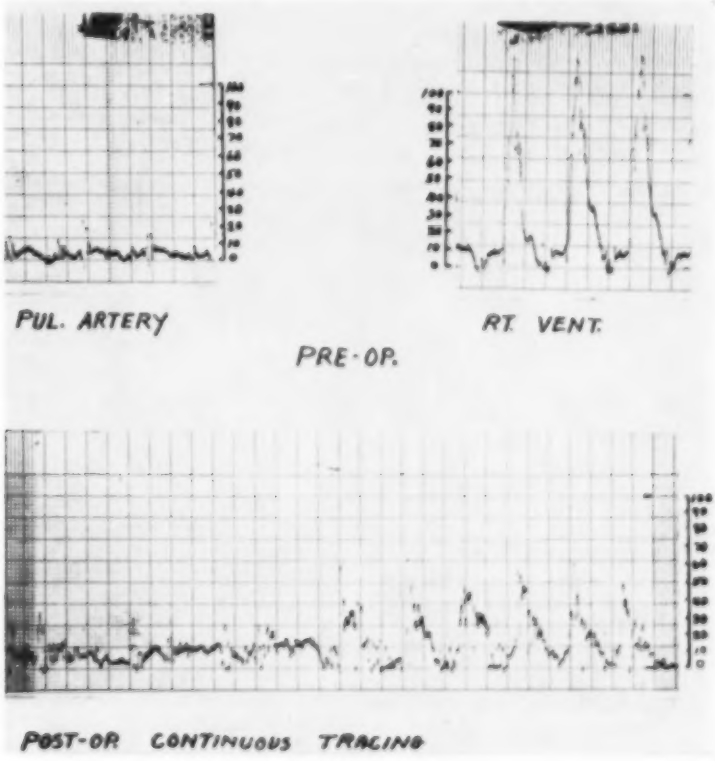

FIGURE 12: Preoperative cardiac catheterization curves in pulmonary artery and right ventricle and a postoperative continuous cardiac catheterization curve from pulmonary artery to right ventricle, Case 2.

ever, gallop rhythm was never detected postoperatively. He slowly, progressively improved until discharge from the hospital four weeks following operation. His postoperative cardiac catheterization (Figure 12) shows great reduction of the pressure gradient between the right ventricle and pulmonary artery. His right ventricular pressure is now within normal limits. It is felt that the remaining gradient may be related more to the extreme dilation of the pulmonary artery than to any residual valvular obstruction.

A 25 year old white female and a 5 year old child with pulmonic valvular stenosis have been operated successfully by the same technique with excellent clinical improvement, but no postoperative catheterization studies have been carried out to date. The former patient presented a patent foramen ovale as well. Digital examination of the valve was carried out in the adult following valvulotomy, and it was felt that the valve had been completely opened. It should be noted that the valvulotome used in children is smaller than that used in adults.

The guillotine knife was used on a fifth occasion in an unsuccessful operative case of the tetralogy of Fallot. Hypothermia was used as an adjunct in this case to reduce the patient's metabolic requirements. Probe exploration revealed both infundibular and valvular stenosis. Cardiac arrest developed, and a hurried, emergency infundibular resection, plus opening of one side of the stenosed valve was carried out. Finally, cardiac action was restored to normal rhythm after having passed through several

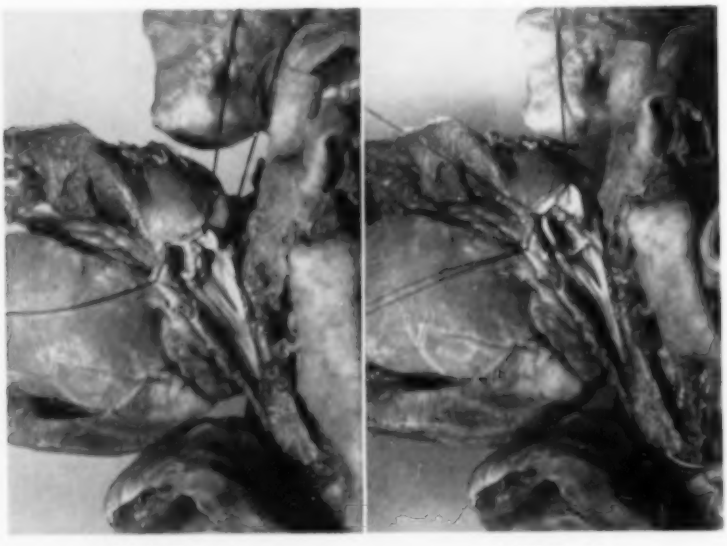

FIGURE 13A FIGURE 13B

Figure 13A: Pulmonic valve viewed from pulmonary artery side showing medial, unilateral incision.—*Figure 13B:* Valve cusps retracted to show incision extending to pulmonic ring.

episodes of ventricular fibrillation. However, the patient expired 12 hours postoperatively with pulmonary edema. Postmortem examination showed that the hurried relief of the infundibular stenosis had resulted in overdoing it (complete relief of the obstruction), hence, in creation of a great left-to-right shunt which caused the pulmonary edema. However, examination of the valve revealed that the unilateral cut with the guillotine knife had extended to the pulmonic ring (Figure 13).

SUMMARY

Experiences with 115 cases of congenital pulmonary stenosis treated by the direct operative approach of Brock and the operative mortality incurred are reported. A new rongeur for resection of infundibular stenosis is presented. A guillotine-type knife is also presented which has been used successfully to alleviate pulmonary valvular stenosis by a blind "closed technique." It is believed that these two technical aids will render it possible more safely and more adequately to relieve the pulmonic obstruction associated with the tetralogy of Fallot and isolated forms of pulmonic stenosis without the necessity for employing the more complicated and inherently more dangerous open techniques.

RESUMEN

Se refieren las experiencias resultantes de 115 casos de stenosis pulmonar congénita tratada por las vía directa de Brock y la mortalidad en que se incurre. Se presenta una nueva gubia para resección de la estenosis infundibular. También se presenta un cuchillo en forma de guillotina que ha sido satisfactorio para corregir la estenosis valvular de una "técnica ciega cerrada."

Se cree que estas dos técnicas harán posible corregir la obstrucción pulmonar asociada a la tetralogía de Fallot y con las formas deestenosis pulmonar sin necesidad de usar las técnicas abiertas que son más complicadas y por sí más peligrosas.

RESUME

Les auteurs rapportent 115 observations de rétrécissement pulmonaire congénital, traité par l'opération directe de Brock, et ils fournissent une statistique de leur mortalité opératoire. Ils présentent un nouvel appareil qui permet la résection du rétrécissement de i'infundibulum. Ils présentent également un bistouri à guillotine, grâce auquel on a pu supprimer le rétrécissement des valvules pulmonaires, en opérant à l'aveugle. Ils pensent que ces deux instruments permettront un traitement plus sûr et mieux adapté des rétrécissements pulmonaires isolés ou associés à la tétralogie de Fallot. Ainsi ne sera-t-il plus nécessaire d'utiliser les techniques à ciel ouvert plus compliquées et par conséquent plus dangereuses.

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Lung Biopsy in Diffuse Pulmonary Disease*

PETER A. THEODOS, M.D., F.C.C.P., FRANK F. ALLBRITTEN, JR., M.D.
and ROBERT L. BRECKENRIDGE, M.D.

Philadelphia, Pennsylvania

Diffuse bilateral pulmonary disease frequently presents a difficult diagnostic problem. The increased use of chest roentgenograms and the emphasis on photofluorographic surveys has accentuated this problem by bringing to attention many such cases, generally asymptomatic or with minimal complaints. The diagnosis can often be determined from history, physical examination, skin tests, peripheral lymph node biopsy, examination of bronchial aspirate, sputum and other studies. However, diagnosis of these diffuse lesions by such methods is generally difficult, time consuming and expensive, and a definitive diagnosis is frequently not obtained. A simple ingenious technique for obtaining a representative specimen of lung tissue for histologic study under direct visualization through a small incision has been devised by Klassen and his group.[1] The present report summarizes our experience with the use of this method in 50 patients.

Surgical Method

The biopsy is done with local infiltration anesthesia. This anesthetic is chosen for many of the patients requiring a diagnostic lung biopsy are respiratory and cardiac cripples. The site of biopsy is chosen over the areas showing the greatest evidence of disease roentgenographically. An anterior incision is preferred if the disease is homogenously diffuse for the greater width of anterior interspaces provide a larger space through which to work. Before the incision is made the face mask is placed and the patient is asked to breath against positive pressure in order to acquaint him with what will be required during the procedure. The mask is then removed.

An incision is made over the site selected. The muscles are split in the line of their fibers down to the interspaces. The intercostal muscles are incised to the parietal pleura. When the pleura is opened, the lung is grasped with an Allis forceps. The lung is kept expanded by positive pressure oxygen given through a snugly fitting face mask. Various portions of the lung are palpated for a suitable site for biopsy. The site is chosen and is demarcated by mattress cotton sutures. The lung parenchyma is then incised and the section desired removed. Cotton mattress sutures are placed in the lung substance as the dissection proceeds. Multiple specimens can be obtained if required. The site of the biopsy is carefully inspected for bleeding or air leaks and such points are closed with addi-

*From the Departments of Medicine (Dr. Theodos), Surgery (Dr. Allbritten) and Pathology (Dr. Breckenridge) of the Jefferson Medical College and Hospital, Philadelphia, Pennsylvania.

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tional sutures. The lung is then dropped back in the pleural space but is kept expanded by the continued application of positive pressure. No attempt is made to close the intercostal muscle bundle; the overlying muscle, subcutaneous tissue, and skin are closed anatomically. The sutures are removed on the fifth or sixth post-operative day.

TABLE I

Diagnosis of diffuse pulmonary lesions as established by lung biopsy in 50 patients

Histologic Diagnosis	Number of Patients
Anthracosiliconis	6
Siliconis	2
Anthracosis	4
Asbestosis	3
Berylliosis	2
Tuberculosis	2
Sarcoidosis	7
Diffuse interstitial fibrosing pneumonitis	4
Non-specific pulmonary fibrosis	6
Non-specific pulmonary fibrosis and emphysema	3
Emphysema	3
Atelectasis	2
Non-specific chronic pneumonitis	2
Chronic passive congestion	2
Cystic disease	1
Pulmonary lipiodosis	1

Results

Table 1 shows the histological diagnosis as reported in the 50 patients comprising this series. In 27 (54 per cent), a specific diagnosis was established by histological and bacteriological study of the biopsy specimen (Table 2). In 26 of these 27 patients, the pre-operative diagnosis was confirmed by biopsy. In eight additional patients the pre-operative impression was also confirmed by biopsy although a specific entity was not identified, (Cases 8, 15, 18, 19, 23, 24, 26, 41).

The following are the case reports of eight representing a cross section of some of the possible diagnoses that can be made with this method.

Case 8: G. C., a 45 year old white male was first seen because of progressive exertional dyspnea for the past two years, moderate cough and occasional wheezing. His work was that of a weaver in an asbestos factory for 10 years. Physical examination revealed evidence of marked emphysema. A roentgenogram (fig. 1A) revealed a diffuse increase in lung markings with peribronchial fibrosis at both bases. Serial films had shown negative findings five years previously and beginning changes at the bases two years before admission. Physiological studies revealed a vital capacity of 2125 cc (48 per cent of predicted normal), maximal breathing capacity of 84 L/min (68 per cent of normal) and residual air of 1836 cc representing 46 per cent of total lung volume. These findings were indicative of an advanced degree of emphysema and pulmonary function impairment. At operation the right pleural space was found

to be free. The lung was grossly nodular and showed a moderate, diffuse, black pigmentation. The biopsy specimen revealed asbestosis, pulmonary fibrosis and emphysema (fig. 1B). The operation was well tolerated despite the advanced disability and no post-operative complication occurred.

Case 5: R. B., a 65 year old white male was referred because of progressive dyspnea for the past three years, moderate cough and expectoration. There was a complex industrial exposure, basically that of working in and around sand mines for eight to 16 years and in a cement factory using limestone for 30 years. Clinically he revealed advanced emphysema and advanced pulmonary function impairment. Roentgenograms (fig. 2A) revealed a moderate degree of linear and nodular fibrosis scattered throughout both lungs consistent with the diagnosis of atypical second stage silicosis. Many previous opinions had been expressed as to the roentgenographic diagnosis ranging from non-specific bronchitis to early pneumoconiosis. Industrial compensation had been denied on the assumption that the pulmonary changes were not due to silicosis. At operation the right lung was found free in the pleural space. There was firm nodulation throughout the upper lobe and a specimen was obtained from this region. Histologic study (fig. 2B) revealed numerous silicotic nodules. No complications occurred.

Case 10: V. B., a 34 year old white female was referred for hospitalization with the diagnosis of far advanced, active, pulmonary tuberculosis. This diagnosis had

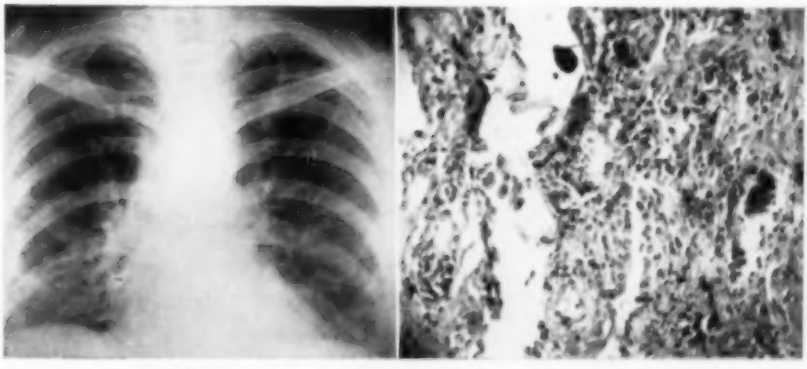

FIGURE 1A FIGURE 1B

Figure 1—Case 3: Asbestosis. *A.* Roentgenogram showing diffuse increase in lung markings with peribronchial fibrosis in lower lobes. *B.* Photomicrograph showing moderate interstitial fibrosis, prominent bronchiolar lining epithelium and the characteristic asbestos bodies. (× 200).

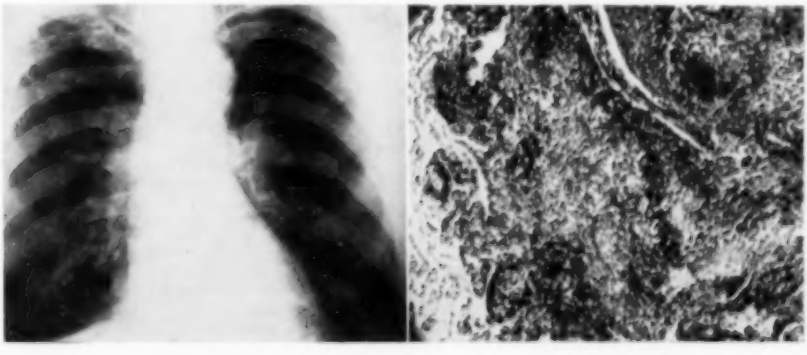

FIGURE 2A FIGURE 2B

Figure 2—Case 5: Silicosis. *A.* Roentgenogram showing linear and nodular fibrosis. *B.* Photomicrograph showing silicotic nodules coalescing to form a large, irregular, darkly pigmented nodule. (× 50).

TABLE II

Diagnostic Effectiveness of Biopsy of Diffuse Pulmonary Lesions in 50 Patients

Case		Preoperative Clinical Diagnosis	Biopsy Diagnosis	Specific Diagnosis Established By Biopsy	Preoperative Diagnosis Confirmed By Biopsy
1.	P.N.	Borderline anthracosilicosis	Anthracosis	No	No
2.	R.B.	Borderline anthracosilicosis	Anthracosis	No	No
3.	G.C.	Asbestosis	Asbestosis of lung with pulmonary fibrosis and emphysema	Yes	Yes
4.	G.K.	Tuberculosis	Fibro-caseous tuberculosis and anthracosis of lung	Yes	Yes
5.	R.B.	Silicosis	Silicosis	Yes	Yes
6.	D.C.	Resolving sarcoidosis	Atelectasis, no sarcoid	No	No
7.	A.S.	Anthracosilicosis	Anthracosilicosis	Yes	Yes
8.	J.V.	Chronic pneumonitis nature undetermined	Chronic inflammatory disease of lung with congestion and fibrosis. No tuberculosis	No	Yes
9.	J.R.	Sarcoid	Sarcoidosis	Yes	Yes
10.	V.B.	Sarcoid	Sarcoidosis	Yes	Yes
11.	G.J.	Pulmonary fibrosis, nature undetermined	Diffuse interstitial fibrosing pneumonitis	Yes	No
12.	M.M.	Cystic disease	Cystic disease	Yes	Yes
13.	B.H.	Early sarcoid	Vesicular emphysema, anthracosis	No	No
14.	S.B.M.	Silicosis	Silicosis	Yes	Yes
15.	D.W.	Pulmonary fibrosis, nature undetermined	Diffuse fibrosis of lung with emphysema	No	Yes
16.	E.B.	Sarcoid	Sarcoidosis	Yes	Yes
17.	P.R.	Sarcoid	Pulmonary granuloma consistent with tuberculosis or sarcoid	Yes	Yes
18.	L.H.	Pulmonary fibrosis and emphysema	Vesicular emphysema	No	Yes
19.	J.B.	Pulmonary fibrosis and emphysema	Pulmonary emphysema, fibrosis and anthracosis	No	Yes
20.	C.R.	Pulmonary fibrosis, non-specific	Atelectasis, slight	No	No
21.	G.W.	Silicosis	Emphysema with interstitial fibrosis	No	No
22.	A.H.	Sarcoid	Non-specific fibrosis, no emphysema and atelectasis	No	No
23.	I.H.	Pulmonary fibrosis, non-specific	Non-specific interstitial fibrosis with emphysema	No	Yes
24.	J.C.	Pulmonary fibrosis, non-specific	Pulmonary fibrosis and anthracosis	No	Yes

TABLE II—(*Continued*)

Diagnostic Effectiveness of Biopsy of Diffuse Pulmonary Lesions in 50 Patients

Case		Preoperative Clinical Diagnosis	Biopsy Diagnosis	Specific Diagnosis Established By Biopsy	Preoperative Diagnosis Confirmed By Biopsy
25.	W.L.	Anthracosilicosis	Anthracosilicosis	Yes	Yes
26.	S.McQ.	Pulmonary fibrosis, non-specific	Pulmonary fibrosis, non-specific	No	Yes
27.	E.W.	Anthracosilicosis	Emphysema and anthracosis	No	No
28.	J.T.	Asbestosis	Asbestosis	Yes	Yes
29.	A.F.	Pulmonary changes secondary to rheumatic fever	Chronic passive congestion	No	No
30.	A.S.	Borderline anthracosilicosis	Anthracosis with chronic passive congestion of lung	Yes	Yes
31.	E.M.	Sarcoidosis	Chronic granuloma compatible with sarcoidosis	Yes	Yes
32.	L.C.	Sarcoidosis	Sarcoidosis	Yes	Yes
33.	C.S.	Sarcoidosis	Chronic passive congestion, fibrous pleuritis	No	No
34.	T.G.	Asbestosis	Asbestosis	Yes	Yes
35.	T.B.	Anthracosilicosis	Anthracosis	No	No
36.	P.McA.	Anthracosilicosis	Anthracosilicosis	Yes	Yes
37.	D.J.	Berylliosis	Berylliosis	Yes	Yes
38.	I.S.	Miliary tuberculosis	Chronic granulomatous inflammation of lung compatible with tuberculosis	Yes	Yes
39.	D.L.J.	Diffuse interstitial fibrosing pneumonitis	Diffuse interstitial fibrosing pneumonitis	Yes	Yes
40.	R.G.	Pulmonary eosinophiliosis	Non-specific fibrosis	No	No
41.	M.T.	Pulmonary fibrosis and emphysema	Pulmonary fibrosis with emphysema, etiology undetermined	No	Yes
42.	M.S.	Anthracosilicosis	Anthracosilicosis	Yes	Yes
43.	M.C.	Pneumolipiodosis	Calcified lipiodol in Alveoli	Yes	Yes
44.	E.W.	Sarcoidosis	Fibrosis of lung with atelectasis	No	No
45.	G.R.	Diffuse interstitial fibrosing pneumonitis	Diffuse interstitial fibrosis of lung	Yes	Yes
46.	J.T.	Sarcoidosis	Granuloma of lung consistent with sarcoidosis	Yes	Yes
47.	T.D.	Pulmonary fibrosis and emphysema	Interstitial pneumonitis with focal areas of atelectasis	No	No
48.	H.C.	Sarcoidosis	Focal areas of emphysema and pneumonitis	No	No
49.	B.Y.	Anthracosilicosis	Anthracosilicosis	Yes	Yes
50.	G.F.	Berylliosis	Berylliosis	Yes	Yes
				27 (54%)	34 (68%)

been made independently by two radiologists. There was a history of progressive dyspnea for the past two years with slight cough, expectoration, marked fatigue and weight loss of 28 pounds over the past six months. Clinical examination was essentially negative except for an enlarged spleen. The roentgenogram (fig. 3A) revealed a diffuse nodular fibrotic infiltration throughout both lungs with coalescence in the second right anterior interspace. On day of admission to the hospital pulmonary biopsy was done. The pleural space was free of adhesions and the lung revealed fine nodulation throughout the upper lobe. The hilar lymph nodes were enlarged and firm. Histologic examination revealed numerous tubercles of the solid type with only slight inflammatory reaction. The granulomas did not coalesce and contained giant cells of the foreign body type. Asteroid bodies were present in a few of the giant cells. The diagnosis was Boeck's sarcoid (fig. 3B). Subsequent studies revealed a negative tuberculin test and no acid-fast bacilli in the sputum. Routine bacteriologic study of the tissue proved sterile. She was discharged on the third post-operative day to return to her occupation as a secretary.

Case 11:[3] G. J., a 57 year old white male was admitted to the hospital because of marked dyspnea for three years and congestive heart failure for one year. There

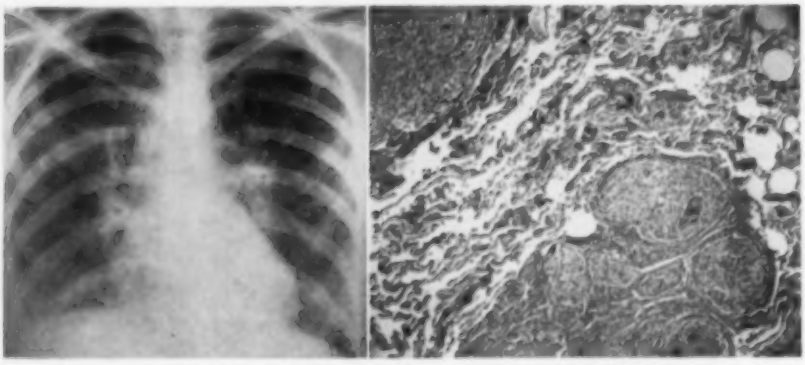

FIGURE 3A FIGURE 3B

Figure 3—Case 10: Boeck's Sarcoid of Lung. *A.* Roentgenogram showing diffuse nodular fibrotic infiltration with coalescence in the second right anterior interspace. *B.* Photomicrograph showing hard tubercules with only a mild inflammatory reaction and containing an occasional multinucleated giant cell of the foreign body type. (× 50).

FIGURE 4A FIGURE 4B

Figure 4—Case 11: Diffuse fibrosing interstitial pneumonitis (Hamman-Rich). *A.* Roentgenogram showing diffuse mottled infiltration, marked emphysema, left ventricular hypertrophy and prominent pulmonary conus and arteries. *B.* Photomicrograph. Note the marked interstitial fibrosis, the focal areas of obliteration of the alveolar lumens, the septal-cell proliferation, the fibrinous masses, and the minimal inflammatory reaction except for focal collections of lymphocytes. (× 50).

was a history of using sand for plate graining and for grinding steel balls over a period of six years. He stated that he had been bled once *every* 21 years as a child for "high blood." Clinical examination revealed marked cyanosis, orthopnea, clubbing of the fingers, marked emphysema, enlarged liver and edema of the legs. The blood count showed polycythemia (HB 16.5 gms., RBC—5,900,000, hematocrit—59 per cent). An electrocardiogram was consistent with the diagnosis of myocardial damage. A roentgenogram of the chest (fig. 4A) showed a diffuse mottled infiltration throughout both lungs, marked emphysema, left ventricular hypertrophy and a prominent conus and pulmonary arteries. The clinical considerations were early silicosis, pulmonary congestion on a cardiac basis, fibrosis from Ayerza's disease and fibrosis from the polycythemia. At operation the lung was extremely dark and finely nodular throughout the area that could be palpated. Pulmonary biopsy revealed diffuse fibrosing interstitial pneumonitis (fig. 4B), a specific pathological entity, first reported by Hamman and Rich.[2] The prominent histologic features were: Marked interstitial fibrosis, focal areas of obliteration of the alveolar lumens, septal-cell proliferation, scattered hyaline membranes and fibrinous masses, and a minimal inflammatory reaction with collection of lymphocytes and plasma cells. No post-operative complications occurred except slight subcutaneous emphysema.

Case 14: S. B. M., a 54 year old white male was first seen in March 1952 with a two year history of dyspnea, slight cough, moderate weight loss and wheezing. He had been a brick mason for 26 years. Tuberculosis was suspected in 1942 and he had spent five months in bed. At time of admission to the hospital clinical and roentgenographic (fig. 5A) examinations revealed a moderate degree of emphysema with diffuse coalescent nodular infiltration in the upper one-half of both lungs, enlarged heart and advanced degree of pulmonary function impairment. The cause of the fibrosis was obscure but silicosis was suspected. He was discharged after a course of intermittent positive pressure breathing which produced considerable symptomatic relief. He was readmitted in September, 1952 when his condition became progressively worse. At this time he was in obvious congestive heart failure. A diagnosis of pulmonary tuberculosis and silicotuberculosis had been made in the interim by other consultants. Roentgenograms taken during the present admission revealed no change in the pulmonary lesions. It was decided to perform a biopsy to determine the exact cause of the fibrosis. At time of operation approximately two liters of fluid were found in the right pleural space. Despite careful pre-operative fluoroscopic and roentgenographic examinations the presence of the fluid had not been suspected. There was little evidence of disease peripherally, the maximum amount of disease being in the hilar areas. Several biopsy specimens were taken which revealed silicosis on microscopic examination (fig. 5B). The operation was initially well tolerated but a residual minimal hydropneumothorax aggravated the existing pulmonary insufficiency and the patient expired suddenly on the third post-operative day, apparently from cardiac failure. The essential gross post-mortem findings were: massive right pleural effusion with blood staining secondary to the operative procedure; shift of the heart and mediastinum to the left; silicosis of the lungs, hilar and mesenteric lymph nodes; cardiac dilitation; bilateral chronic fibrous pleuritis; and arteriosclerosis of the kidneys. Histologically the silicosis was associated with atelectasis, emphysema and chronic pleuritis.

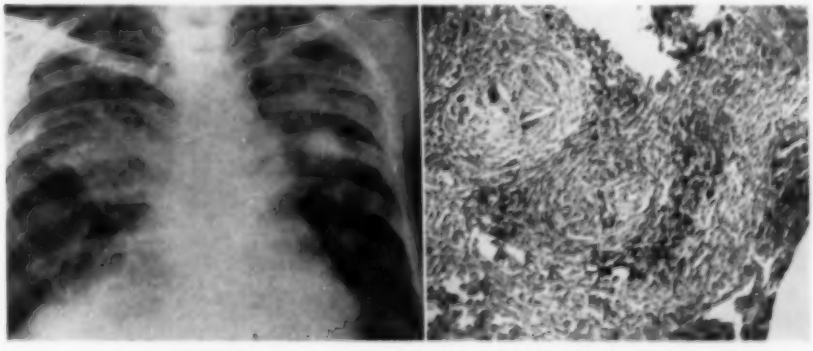

FIGURE 5A FIGURE 5B

Figure 5—Case 14: Silicosis. *A.* Roentgenogram showing diffuse coalescent nodular infiltration in upper half of both lungs. *B.* Photomicrograph showing silicotic nodulation. The two nodules contain multinucleated giant cells surrounding crystal-like slits. The fibrosis and pigmentation is prominent around the nodules. (× 100).

Case 15: D. W., a 63 year old white male was referred because of a previous diagnosis of pulmonary fibrosis. His complaints were marked dyspnea for one year with severe cough and expectoration for two years and edema of the ankles for two weeks. Two years previously there was a history of work with iron dust for 10 months but no other industrial exposure. Clinical examination revealed cyanosis of the lips, clubbing of the fingers, roughened breath sounds and moist rales at both bases. The degree of emphysema appeared to be slight but there was obvious congestive heart failure and a far advanced degree of pulmonary function impairment. An electrocardiogram revealed myocardial damage and cor pulmonale. Pulmonary function studies revealed vital capacity 34.4 per cent of predicted normal and maximal breathing capacity 55.5 per cent of predicted normal. Roentgenogram of the chest (fig. 6A) disclosed diffuse fibrosis of both lungs radiating out from the hilar regions with little emphysema. The nature of the fibrosis and its relationship to the exposure to iron dust were the points at issue. At time of biopsy the entire right lung appeared abnormal. There was a diffuse increase in the density of the lung without palpable nodules. The histologic specimen (fig. 6B) revealed chronic, non-specific fibrosis with emphysema and hyaline masses within the alveoli. The operation was well tolerated despite the extreme dyspnea. Slight residual pneumothorax which aggravated the dyspnea was present for several days but no other ill effects were noted.

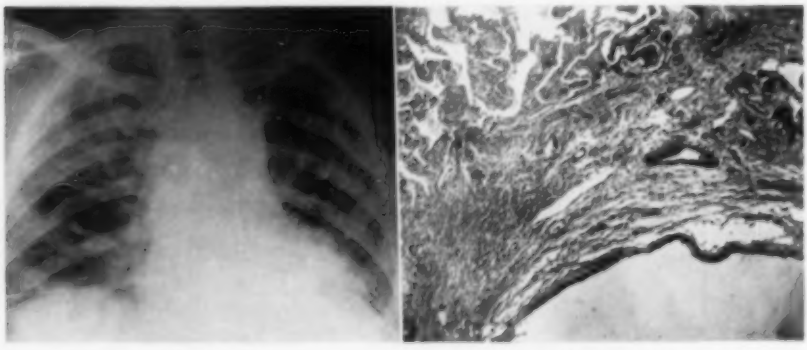

FIGURE 6A FIGURE 6B

Figure 6—Case 15: Pulmonary fibrosis, non-specific. *A.* Roentgenogram showing diffuse fibrosis. *B.* Photomicrograph showing a moderate infiltration of plasma cells and lymphocytes, emphysematous blebs and organized hyaline masses within the alveoli. (× 50).

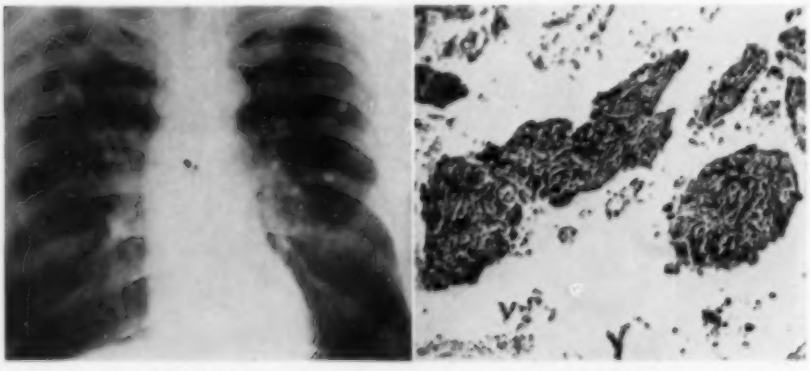

FIGURE 7A FIGURE 7B

Figure 7—Case 35: Anthracosis. *A.* Roentgenogram showing scattered nodulation. *B.* Photomicrograph. The two nodules represent thickened interstitial tissue containing a large amount of black pigment and fibrous tissue. The specific silicotic nodule was not demonstrated. (× 100).

Case 35: T. B., a 53 year old soft coal miner who spent 30 years in the mines was referred because of dyspnea of four years duration. Physical examination was essentially negative but the roentgenogram (fig. 7A) showed a fine nodulation throughout both lung fields. The clinical impression was early anthracosilicosis. At operation the lung was found to be granular without sizable nodules. Histologic examination (fig. 7B) revealed anthracosis but no silicosis. A moderate degree of subcutaneous emphysema developed which reabsorbed spontaneously.

Case 38: I. S., a 37 year old female was admitted to the Jefferson Medical College Hospital because of persistent fever following delivery of a normal child. Prior to delivery a photofluorogram taken in the maternity out-patient department was negative. A roentgenogram (fig. 8A) taken two days post-partum revealed a diffuse, soft, mottled infiltration in both lungs. Aspiration of amniotic fluid was entertained as a possible cause. Failure to improve under antibiotic therapy suggested miliary tuberculosis. This was confirmed by a biopsy. The histological sections showed a chronic granulomatous inflammation of the lung compatible with tuberculosis (fig. 8B). Appropriate chemotherapy was instituted. A prompt drop in temperature occurred with improvement of her general condition.

Discussion

Biopsy of diffuse pulmonary lesions by the method described affords the means of establishing a rapid, accurate diagnosis in many cases which would normally defy diagnosis. Prior to the development of this technique by Klassen, direct examination of lung tissue in the living was possible only by exploratory thoracotomy, or by needle aspiration biopsy. The usual thoracotomy is time consuming, carries a considerable post-operative morbidity and its use is frequently not possible in the presence of marked pulmonary insufficiency. Needle aspiration biopsy of the lung is hazardous and the amount of tissue obtained is frequently insufficient to permit adequate histological, bacteriological or chemical study. These inadequacies are obviated by the present method of biopsy.

Lung biopsy has its greatest efficacy in diffuse lesions such as those seen in the pneumoconioses, sarcoidosis, metastatic malignancy and tuberculosis where a specific pathological entity is present. In these conditions the diagnosis can be readily established since the pathology is sufficiently well-defined to permit accurate diagnosis. In the presence of non-specific

FIGURE 8A

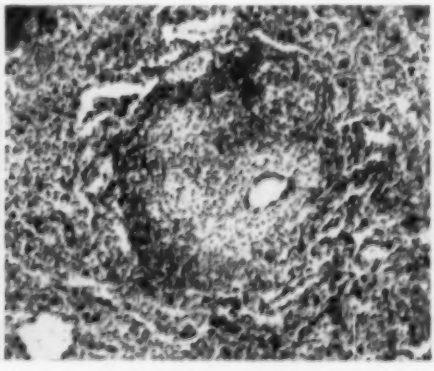

FIGURE 8B

Figure 8—Case 38: Miliary tuberculosis. *A*, Roentgenogram showing diffuse, soft, fine motted infiltration. *B*, Photomicrograph showing chronic granulomatous infiltration of tuberculosis.

conditions such as fibrosis or pneumonitis, however, the biopsy will not result in a specific diagnosis, because none exists (Cases 8, 15, 23, 33, 41). Even at autopsy it is sometimes impossible to determine the cause of the pulmonary changes in these diseases. In addition, when emphysema is a major factor (Cases 1, 2, 18, 19), the pathologic processes may be too centrally located to be reached by peripheral biopsy. The emphysematous changes are generally greater over the anterior aspects of the upper lobes, which are the usual sites for the biopsy. In this series of patients, no specific pathological diagnosis was possible in 46 per cent. Biopsy served only to rule out the presence of a definite disease entity in these patients. Appreciation of the high prevalence of non-specific fibrosis and emphysema in clinical practice should make the use of lung biopsy more selective.

It is probable also that lung biopsy will permit a more specific diagnosis if microincineration and spectrophotometry can be carried out upon the biopsy specimens. The procedure allows for multiple pieces of tissue to be obtained easily but special laboratory techniques and equipment are required for these special studies.

In forensic medicine lung biopsy is of great value. The necessity of determining whether or not the changes in the roentgenogram of a patient's chest have resulted from exposure to injurious dusts is frequently a problem. In cases 5, 7, and 14, the diagnosis of silicosis had been rejected on the basis of atypical roentgenographic findings but the presence of silicosis was readily established by biopsy and compensation justifiably sought. Because silicosis was absent in the biopsy of Case 4 claim for industrial compensation was dropped by the patient.

With the establishment of a specific diagnosis the question of treatment and prognosis can be settled more readily. Prompt institution of intensive chemotherapy in the patient with miliary tuberculosis (Case 38) resulted from early recourse to lung biopsy. In Case 10 the patient was on the verge of terminating her employment as a secretary because of the diagnosis of tuberculosis until the biopsy revealed the presence of sarcoid. Similarly several patients (Cases 1, 2, 27, 35) were advised to continue their usual work when the biopsy revealed an absence of silicosis.

The use of this procedure as a diagnostic aid shortens the period of hospitalization and lessens the cost. Case 16 may be cited as an example. The patient had been hospitalized on a general medical ward for a period of 22 days at an estimated cost of 535 dollars. Extensive diagnostic studies had suggested the diagnosis of sarcoid but this could not be proved. Hospitalization for lung biopsy took four days, cost 134 dollars, and the specific diagnosis of sarcoid was established. The average hospital stay for the biopsy analysis was five days.

Occasionally the establishment of a specific diagnosis through biopsy may cause one to overlook the possibility of associated conditions. Such occurred in Case 46. The patient was biopsied because of diffuse infiltrations in the lungs and a diagnosis of sarcoid was made. Several months later he coughed up a plug of material which was found to be malignant.

Bronchogenic carcinoma was proved on bronchoscopy and resection carried out successfully. It was obvious that both sarcoid and unrecognized malignancy coexisted in this patient at time of biopsy.

The operation was well tolerated although death occurred after lung biopsy in one patient who was in heart failure preceding the operation. The post-operative complications are summarized in Table 3. Subcutaneous

TABLE III

Complications in biopsy of diffuse pulmonary lesions in 50 patients

Histologic Diagnosis	Number of Patients	Subcutaneous Emphysema	Local Reaction in Lung	Pneumothorax	Intra-Pleural Hemorrhage	Pleuritis	Death
Anthracosilicosis	6	2	1	1			
Silicosis	2	1		1	1		1
Anthracosis	4	2					
Asbestosis	3	1					
Beryliosis	2	1					
Tuberculosis	2						
Sarcoidosis	7	1		2		1	
Diffuse interstitial fibrosing pneumonitis	4	1					
Non-specific pulmonary fibrosis	6	1		1			
Non-specific pulmonary fibrosis and emphysema	3	1					
Emphysema	3	1	1				
Other	8	1	1				
Totals	50	13 (26%)	3 (6%)	5 (10%)	1 (2%)	1 (2%)	1 (2%)

emphysema of varying degrees was present in 13 patients (26 per cent), a residual pneumothorax was present in five (10 per cent) and a local parenchymal reaction at site of biopsy in three (6 per cent). A significant pleural reaction was seen in only one. All of these complications promptly subsided spontaneously. Death in the one patient occurred three days postoperatively from cardiac failure secondary to pulmonary insufficiency. A small marginal pneumothorax developed after the biopsy and this probably aggravated a failing heart. In general, however, the risk from this procedure is slight and can safely be applied in the presence of extreme pulmonary disability. In four such patients (Cases 7, 11, 15, 18) the operation was well tolerated and produced no aggravation of existing dyspnea. The fortuitous obliteration of the pleural space will greatly add to the safety of the procedure as collapse of the lung will not occur. To our knowledge no dissemination of lesions or wound infection has occurred.

SUMMARY AND CONCLUSIONS

The use of lung biopsy under local anesthesia for investigation of diffuse pulmonary lesions in 50 patients is presented. A specific diagnosis of the

pulmonary lesion was possible in 27 of the 50 by histologic study of the biopsy tissue. The procedure was well tolerated and produced minimal complications except in one case which ended fatally. It has its greatest applicability in those diseases where a specific pathological entity is present. In general it affords a rapid accurate method for the diagnosis, determination of the medico-legal status, and determining the prognosis of diffuse pulmonary lesions. Eight cases illustrating the value of this procedure are described in detail.

SUMARIO Y CONCLUSIONES

Se presenta el uso de la biopsia pulmonar bajo anestesia local para la investigación de lesiones pulmonares difusas. Fué posible-hacer un diagnóstico específico de la lesión en 27 de las 50 biopsias estudiadas histológicamente. El procedimiento fué bien tolerado y produjo únicamente complicaciones mínimas, a excepción de uncaso que terminó fatalmente. Tiene su mayor aplicabilidad en aquellas enfermedades en que está presente una entidad patológica específica. En general, aporta un método acucioso y rápido para el diagnóstico, para la determinación del estado médico-legal y determina el promóstico de lesiones pulmonares difusas. Se describen en detalle ocho casos que ilustran el valor de este procedimiento.

RESUME

Les auteurs présentent leurs conclusions sur l'empoli de la biopsie pulmonaire sous anesthésie locale chez 50 malades, atteints de lésions pulmonaires diffuses. L'étude histologique de la biopsie permit le diagnostic de la lésion chez 27 d'entre eux. Cette investigation fut aisément tolérée et n'entraina que des ennuis mineurs, sauf dans un cas dont l'évolution fut fatale. Elle a donné les meilleurs résultats dans les cas où l'affection correspondait bien à une entité anatomo-pathologique précise. D'une façon générale, elle permet d'accélérer la détermination du diagnostic, d'éclairer le problème médico-légal, et de faciliter l'établissement du pronostic dans les cas de lésions pulmonaires diffuses. L'auteur pour illustrer l'intérêt de cette technique rapporte huit observations détaillées.

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Prophylaxis Against Air Embolism from Puncture of the Lungs and Pleurae*

With special reference to refills of artificial pneumothorax

GÖSTA BIRATH, M.D., F.C.C.P.
Gothenburg, Sweden

One of the most dramatic accidents in the treatment of pulmonary disease is air embolism, which may complicate all intrathoracic interventions, even the simplest needling procedures.

The frequency figures reported for air embolism vary greatly. The complication appears to have been fairly common in the early years of artificial pneumothorax, presumably due to the higher intrathoracic pressures then employed. Gradually the frequency has declined. Figures from the 1920's and 30's range from 0 to 11 cases per 10,000 refills.[1] The fatality rates vary between 0 and 3 per 10,000 refills. With the exception of the mortality, the reported frequencies must be regarded as minimum figures, since air embolism in many cases undoubtedly has been given such interpretations as syncope and pleural shock.

According to the type of vessel in which the air lodges, it is usual to differentiate between *venous* and *arterial* emboli. The prognosis for those two forms of embolus is quite different. While in a horse 1 ml. of air in the carotid artery can produce unconsciousness, up to 20 liters of air injected intravenously may cause no noteworthy disturbance. Nemec injected 10 ml. of air into his own veins without experiencing any symptom.

Air emboli which complicate such measures as exploratory puncture of the thorax or pneumothorax refills are practically always of arterial type.

The most common form of air embolus in association with pneumothorax and with all other thoracic punctures, is without doubt due to the negative pressure in the pulmonary veins.

Pressures in the Pulmonary Vessels

In 1944 von Bahr[1] showed, by means of animal experiments, that under certain circumstances the pressure in the pulmonary veins becomes negative. Broadly speaking, he found that the pressure in the pulmonary veins above a line drawn through the auricles of the heart was negative, and below that level positive. Von Bahr inserted a 3 cm-long cannula into the ieft auricle of a rabbit and registered the pressure with the animal in various positions. When the normal position was maintained there was a continuous flow of blood through the cannula, which was directed downwards. There was thus positive pressure at the lower end of the cannula, and it was inconceivable that air could be sucked in. When the rabbit was turned so that the cannula lay horizontal, the pressure was still predominantly positive, but on inspiration there was slight sucking in of the column of blood in the cannula lumen, indicating that the pressure in the auricle

*From Renströmska sjukhuset, Gothenburg, Sweden.

was negative. When the animal was turned still further, so that the cannula pointed directly upwards, air was sucked in through it, so that a large air embolus formed. The pressure at the upper end of the cannula, 3 cm above the left auricle, was thus negative. The pressure within the auricle was, of course, also negative. If one thinks of this cannula as representing a pulmonary vein, injury to that vein below the horizontal plane through the left auricle would thus have caused only hemorrhage, while vascular lesion above that plane could have given rise to a suction embolus.

Some personal observations on human subjects tally with these findings. During an adhesion section I noticed that one of the large veins leading from the pleural cupola along the mediastinum and down towards the hilus showed different degrees of filling in different body positions. With the patient supine and the head of the operating table raised about 30 degrees, this vein was totally collapsed. In other words, the pressure within it was lower than in the pleural cavity where, under these circumstances, the pressure may be estimated to be roughly that of the atmosphere. The patient's position was restored to horizontal and the vein was seen to fill during inspiration, when the pressure in the pleural cavity showed some decrease, but collapsed again during expiration with the small increase in pressure which then occurred. When the head of the table was lowered to form an angle of about 20 degrees with the horizontal

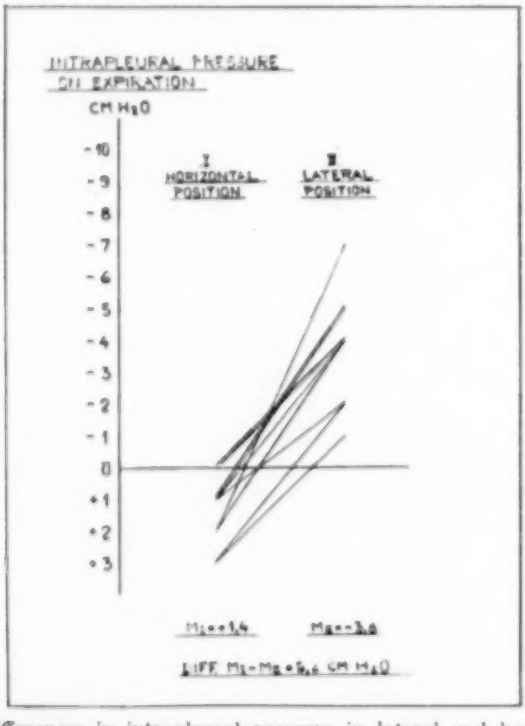

FIGURE 1: Differences in intrapleural pressure in lateral and horizontal position. In above figure, bottom line should read: Diff. M_I-M_{II} = 5, 2 CM H_2O.

plane, the vein was constantly filled. There was thus a positive pressure in the vein during the entire respiratory cycle. The same observations were subsequently made on repeated occasions.

Such findings indicate that von Bahr's interpretation of his experiments was correct. More recent studies with heart catheterization and pressure measurements in the lesser circulation[2] also tally with these earlier deductions. In the recumbent position the capillary pressure on the venous side of the pulmonary circulation was only a few mm Hg, and in the erect position it was still lower. The pressure in the left auricle was considered to be almost nil, although inclining to positive. It was estimated, however, that the laws of hydrostasis must give rise to negative pressure in the venous system above the auricular plane, and below it to positive pressure. In the apices it may be about -18 Hg in erect position.

Under such circumstances it may seem strange that puncture of healthy lung tissue in animal experiments has not produced air embolism. The explanation probably is that when the pressure is negative the veins in healthy tissue can collapse. Such a theory is also to a certain extent supported by the fact that air embolism generally occurs after needling of pathologically altered tissue, with infiltration and induration which may prevent the veins from collapsing. The traumatized vessel therefore remains open and air can easily be sucked in.

Clinical Application

The practical consequence of these circumstances is that for induction and refills of pneumothorax and all other puncture procedures of the lungs or pleurae, the patient should be placed in a position which secures positive pressure in the underlying vessels.

Induction of artificial pneumothorax, therefore, should be performed with the patient supine; a suggested point for insertion of the needle is in the midaxillary line, above healthy lung tissue. In this area there should be at any rate no noteworthy suction effect, except possibly on deep inspiration, which should be avoided during the induction. For the past 10 years I have performed all pneumothorax inductions in this way, and have encountered no practical difficulty. Theoretically it is preferable that the induction needle should be inserted from below, i.e. in the dorsum, but this is not feasible in practice.

Refills of pneumothorax are also carried out with the patient supine. If the lung is well collapsed so that no risk is thereby entailed, the needle is inserted somewhat down towards the base. Nowadays, however, the needle cannot be so introduced in many cases, since selective collapse of the diseased upper lobe is preferred with, if possible, complete expansion of the lower lobe. In such cases the refill is made in the cupola of the pleura close to the scapula, where the lung lies several cm away from the chest wall. The rule concerning puncture over an area with positive pressure in the underlying veins cannot then be applied, as there is risk of injury to the lung. Puncture in the cupola, which does not involve this risk, is the unquestionable choice.

It is very important that if the lung has been accidentally punctured at pneumothorax induction or refill, the patient should remain recumbent for some time. When the lung has been injured this position generally entails less risk of suction air embolus. But if the patient sits or stands up, the negative pressure in the upper lung zones increases and air may be drawn into the vessels. The patient is therefore kept horizontal and moved from the treatment table onto a stretcher, where he must lie for at least one hour. If the puncture has been deeper, so that demonstrable bleeding has occurred, this time must naturally be extended.

Finally it should be pointed out that the differences in intrapleural pressure between the supine and lateral positions are fairly considerable. As figure 1 shows, the pressure generally is about 5 cm higher when the patient lies on his back, a fact which must be taken into account when the position is changed from lateral to supine (Fig. 1).

Even in other punctures of the pleurae and lungs precautions should be taken to ensure that the needle is inserted below the level of the cardiac auricles. If the base of the thorax is to be needled the patient may sit up, but for punctures higher up he must be recumbent, possibly with the head of the table lowered.

Results

The above-described method of procedure has been used by me and my assistants for about 10 years. During this time approximately 60,000 refills have been given 1,200 patients with intrapleural pneumothorax. Air embolism occurred in no case in which these principles were applied. But three air emboli, one of them fatal, were recorded in refills not given as advocated.

The fatal case was a young woman in whom an attempt was made to induce pneumothorax by insertion of the needle at the highest point of the thorax—against the rule. Immediately after the puncture, while the pressure was being measured (—2 +2 cm. of water), she coughed up some blood, lost consciousness and died within a few minutes. Although autopsy showed no air in the heart or the cerebral vessels, a small cerebral air embolism was the only conceivable cause of death. There was total pleural symphysis.

Transient symptoms of air embolism were experienced by two patients who were allowed to descend from the treatment table after the lung had been punctured. The symptoms did not arise until the patients had walked away from the table. One of them had temporary amaurosis, while the other complained only of sweating, faintness and nausea.

No air embolism has occurred in association with extrapleural pneumothorax, despite the high pressures employed (as a rule about + 30 cm of water), and despite the fact that the method at one time was commonly used. Approximately 3.000 such refills have been given.

On the other hand, several cases of air embolism, fortunately without sequelae, have been recorded in connection with extrafascial pneumothorax refills in Semb's thoracoplasty. The rationale of this pneumothorax is that the freed apex of the lung with its attached bows of periosteum should be held down until new rib formation can prevent it from rising again. The frequency of air embolism in Semb's thoracoplasty was about 5 per cent (six or seven patients in about 110 operated cases). Calculated on the number of refills (about 1100) the figure was approximately 0.5 per cent, a remarkably high frequency. These complications

did not occur in the immediate postoperative period, when the pressures were kept low. It is propable that the later high intrathoracic pressures (about +20 +30 cm H_2O) gave rise to a small rupture of a vessel, where air then entered. I believe that this high frequency of air embolism in our cases of extrafascial pneumothorax wasn't a chance phenomenon, since the complication has been very infrequent or absent in the other groups. But the reason for this higher frequency is difficult to determine. Because of this risk we now fill the pleural space after Semb's operation with physiologic saline solution. Since that was introduced we have had no air embolism.

SUMMARY

Pressure in the pulmonary veins above the level of the cardiac auricles appears to be negative. If a vein is injured above that level, there is risk that air may be sucked into it. The risk is particularly great when the lung tissue has undergone changes which lessen the vessel's capacity to collapse. The patient's position for puncture of the chest which may injure the pulmonary parenchyma should therefore ensure that the intervention takes place below the auricular level. If it is known that the lung has been injured, the same position should be maintained for at least one hour afterwards.

RESUMEN

La presión de las venas pulmonares arriba del nivel de las aurículas cardiacas, parece ser negativa. Si una vena es herida, arriba de ese nivel hay el peligro de que ingrese aire aspirado a ella.

El riesgo es particularmente grande cuando el tejido pulmonar ha sufrido cambios que disminuyen la capacidad del vaso para colapsarse.

La posición del enfermo para una punción del tórax que pudiera lesionar el parenquima pulmonar, debe por tanto hacerse de modo que la intervención tenga lugar abajo del nivel de la aurícula.

Si se sabe que el pulmón ha sido lesionado, la misma posición debe mantenerse por lo menos por una hora después.

RESUME

On peut constater que la pression de la veine pulmonaire au-dessus du niveau des oreillettes est négative. Si une veine est lésée au-dessus de ce niveau, on court le risque que soit ainsi provoquée une aspiration d'air. Ce danger est particulièrement important quand les altérations du tissu pulmonaire ont diminué la capacité rétractile des vaisseaux.

Il en résulte que lorsqu'on fait une ponction thoracique qui risque de léser le parenchyme pulmonaire, il est nécessaire de places le malade d'une manière qui la ponction soit jaite au-dessus du niveau des oreilettes. Lorsqu'on s'est aperçu que le poumon a été lésé, il est nécessaire de maintenir le malade dans la même position encore au moins une heure après.

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The Pulmonary Manifestations of Fibrocystic Disease of the Pancreas*

PAUL A. di SANT'AGNESE, M.D.
New York, New York

Cystic fibrosis of the pancreas is an hereditary disease of children characterized by the association of pancreatic deficiency and chronic pulmonary disease. It was briefly recorded in 1936 by Fanconi in Switzerland,[1] but its first complete description was given in 1938 by Andersen in this country.[2] Simultaneously in the same year, two smaller series of patients were reported by two other groups,[3, 4] one in the United States and one in Australia. Pancreatic enzymes are usually, but not always absent at birth; the respiratory involvement has a variable onset from the age of a few weeks to that of several years. Fibrocystic disease of the pancreas occurs equally in all subdivisions of the white race; it is rare in Negroes and has never been found in Mongolians.

It was therefore recognized from its first descriptions[1, 2] that this condition involved both the pancreatic and pulmonary areas. It has recently been shown[5, 6] that yet another organ system, the sweat glands, is consistently affected in this disease; while less commonly hepatic cirrhosis may also be present.[7, 8, 9]

The pathologic lesions in the pancreas and the clinical manifestations of pancreatic deficiency first attracted attention and gave the disease its name. However, a variable degree of bronchial and pulmonary involvement is a virtually constant feature of the disease, usually dominates the clinical picture and determines the fate of the patient.[10, 11]

The basic nature of this widespread disturbance is as yet not known. It has been postulated[12] that mucus secreted in various parts of the body may be abnormal in its physico-chemical characteristics, thus causing obstruction of the pancreatic ducts with consequent dilatation of secretory acini and parenchymal fibrosis. By the same token, failure to remove bronchial mucus secretion leads to obstruction of the air passages and secondary bronchopneumonia. This hypothesis thus gives a reasonable explanation of the pulmonary and pancreatic lesions, but does not account satisfactorily for the electrolyte abnormality of sweat.

There is an accumulating body of evidence to suggest that the secretory activity of many and perhaps all exocrine glands, mucus-producing and others, is affected in this condition. Therefore, it must be recognized that so-called cystic fibrosis of the pancreas is in reality a generalized disease of which the pancreatic and pulmonary lesions are only one expression.

From the Department of Pediatrics, Columbia University and the Babies Hospital. Supported in part by the Robert Bacon Whitney Fund for Cystic Fibrosis of the pancreas, and in part by the Corning Fund for Medical Research.
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I. Respiratory Involvement

In the 15 years from 1939 to 1954, 292 children with cystic fibrosis of the pancreas have been seen at Babies Hospital. Only 21 were hospitalized in the first five years, while 270, an average of 27 new patients per year, have been admitted in the last decade. The increase in the number of cases in the last 10 years is a reflection both of greater recognition of the disease and of the advent of effective antibiotic agents which greatly prolong the life span. In all of these patients pulmonary involvement of varying degree occurred at some time in the course of the illness. In the average case the respiratory disease was severe. In the whole group it accounted for 90 per cent of the 133 deaths.

The age of onset of the pulmonary component varies greatly and ranges from a few days after birth to the age of several years. In the great majority of cases it has its onset between six months and two years of age.

The general pattern of the respiratory disease is strikingly uniform, except for the degree of severity and the duration of single phases.

Initially, following a mild unresolved acute respiratory infection, a dry, non-productive, hacking cough sets in and persists despite treatment for weeks or months. Although the state of nutrition as a consequence of the pancreatic deficiency is variable, the patient frequently does not appear sick at this time. Temperature is within normal limits, on auscultation of the chest a few scattered rales may be heard and little may be seen in roentgenograms except for an increase in bronchovascular markings, and early signs of obstructive emphysema. Respirations are often wheezing, with a prolonged expiratory phase and a diagnosis of so-called "asthmatic bronchitis" is frequently made. The cough may at times become paroxysmal and simulate pertussis. Antibiotics have a beneficial, but only temporary effect on the as yet mild respiratory involvement.

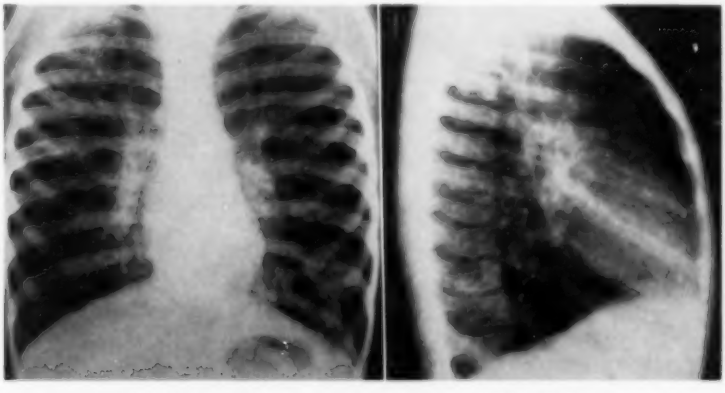
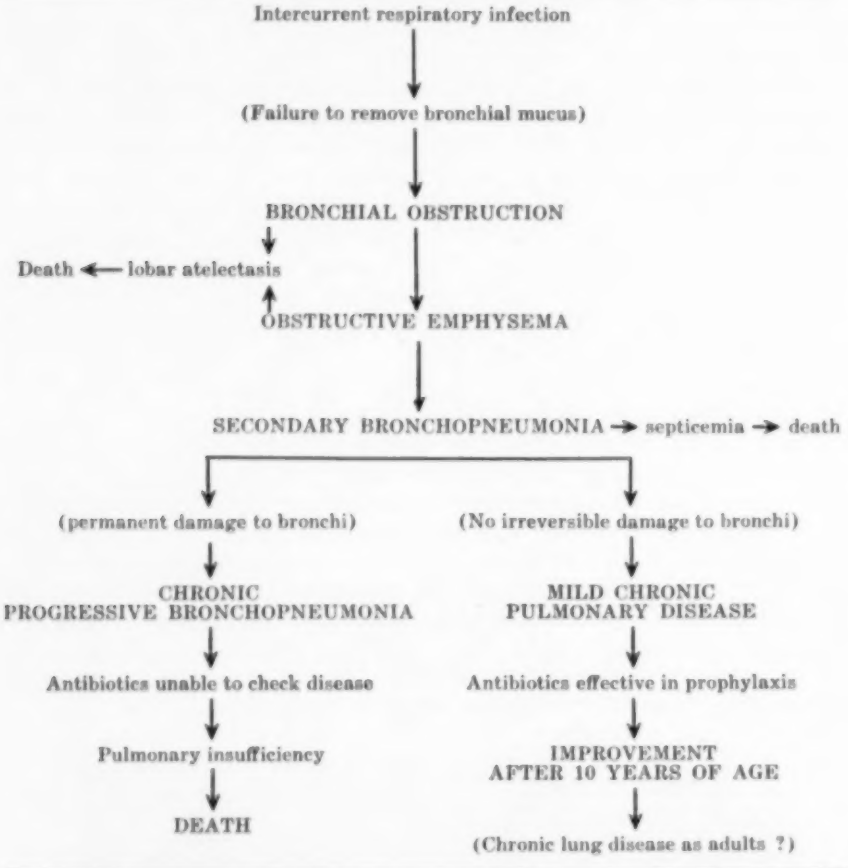


FIGURE 1A FIGURE 1B

Figure 1—A & B: L. N., female, age four years—generalized obstructive emphysema and chronic bronchopneumonia.

TABLE I
RESPIRATORY INVOLVEMENT
IN CYSTIC FIBROSIS OF THE PANCREAS*



*In parenthesis steps that are not proved.

Then suddenly (Table I), following another more severe intercurrent respiratory infection, the failure to remove bronchial secretions leads to widespread and severe bronchial obstruction. The infection, at first mild and perhaps localized to the main divisions of the bronchi, suddenly becomes widespread and severe by invasion of the obstructed air passages down to their smaller subdivisions. The temperature is elevated and the usual laboratory signs of infection are found. Respiratory distress is present to a varying degree and may be severe with cyanosis and air hunger, and the patient is generally quite ill, exhibiting the picture of severe generalized pulmonary infection.

Massive lobar or multilobar atelectasis leading to death may occur at this time.[11] It was also not infrequent in pre-antibiotic days for patients to succumb at this stage because of a spread of the infecting agents to the blood stream. If the patient survives, this cycle can be repeated again on the occasion of subsequent respiratory infections, thus explaining the characteristic course of these patients, punctuated as it is by relatively sudden attacks of markedly increased bronchial obstruction and secondary infection. Any one of these episodes may be fatal.

The physical examination and the roentgen picture are both quite characteristic at this stage. The chest is barrel-shaped and the note on percussion is markedly tympanitic. On auscultation there are scattered rales which clear up temporarily after an episode of paroxysmal coughing. There is gradually increasing clubbing of fingers and toes. On roentgenographic examination the signs of marked obstructive emphysema can be seen (Fig. 1, A & B): Markedly increased antero-posterior diameter of the chest, flattening of diaphragmatic leafs, and a heart which is small in relation to the total thoracic diameter. In addition, the signs of chronic bronchopneumonia are present with a "honey-comb" appearance of the lungs and often marked peribronchial cuffing.

If permanent damage is done to the bronchial wall (Table I), the patients go on to a severe progressive bronchopneumonia, not checked by antibiotics. The x-ray film frequently shows diffuse focal "snowflake" shadows similar to those of disseminated tuberculosis (Fig. 2). Measurements of pulmonary function[13] at this time show the vital and total lung capacity to be greatly reduced, in association with a marked increase in the volume of residual air and the index of intra-pulmonary mixing. There is profound arterial anoxia with marked increase in the CO_2 tension. The

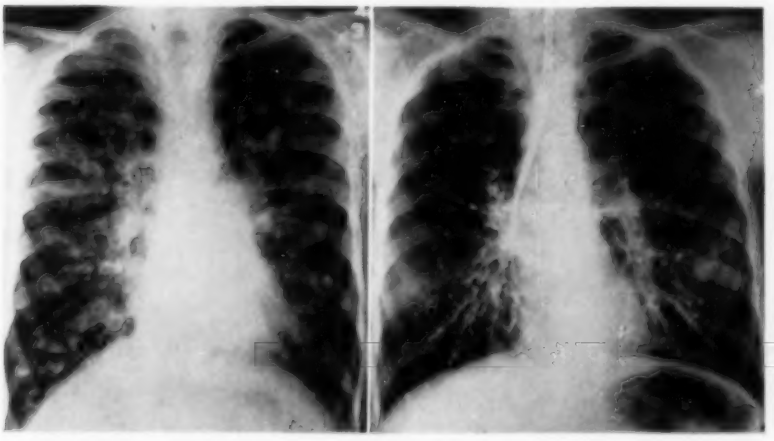


FIGURE 2 FIGURE 3

Figure 2—G. D., male, age 13 years: Severe chronic bronchopneumonia (patient died five months later) ([12]).—*Figure 3*—R. B., female, age 16 years: No evidence of bronchiectasis on bronchography, despite moderately severe bronchopneumonia of many years duration.

distressing picture of pulmonary insufficiency thus develops and eventually leads to death in two or three years on the average. At any time, however, events may take a precipitous turn leading rapidly to fatal termination.

At autopsy in patients who have died of severe chronic bronchial and pulmonary infection, there is almost complete occlusion of the bronchial lumens by extremely thick, tenacious, muco-purulent material. Section of a fresh lung gives a striking picture, with pus welling from every bronchus to form whitish mounds against a fairly homogeneous, pale parenchyma. Multiple lung abscesses are not uncommon and bronchiectasis is a consistent finding.

On nose, throat, sputum cultures and from the lungs at autopsy Staphylococcus aureus hemolyticus is usually recovered.[14] Indeed, the finding of this organism is so consistent as to suggest a possible metabolic factor to explain its striking association with this disease. While staphylococcus was frequently obtained in pure culture in pre-antibiotic days, with the advent of broad-spectrum agents it is not uncommon to find proteus, Pseudomonas aeruginosa, E. coli, and even Candida albicans in the nose and throat cultures or at autopsy.

Fortunately, however, some patients do not have quite as grim an outlook. The pulmonary component of fibrocystic disease of the pancreas

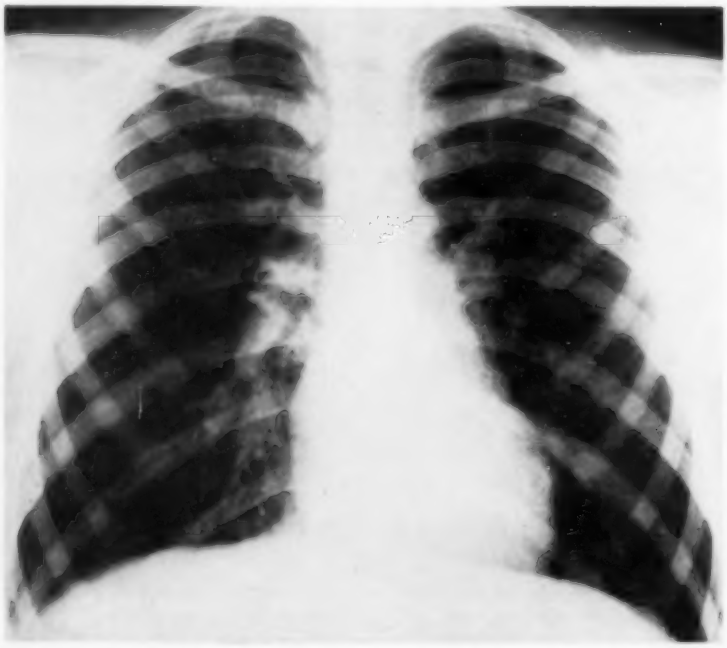


FIGURE 4—V. D.: Normal chest roentgenogram at 14 years. Chronic pulmonary disease of moderate severity from five to 12 years of age. Aureomycin therapy from 11 to 12 years of age.[13]

is an intra-bronchial disease, which does not primarily affect the walls of the respiratory passages. Secondary infection may never cause irreversible damage (Fig. 3), leading only to a mild chronic lung disease which is effectively kept in check by the prophylactic administration of broad-spectrum antibiotics (Table I). If patients reach late childhood without irreversible damage being done to their lungs, they seem to improve and the degree of clearing in the roentgen picture is surprising (Fig. 4). Pulmonary function at this time may be virtually normal.[18] What the future holds for such patients we do not know, but suspect that at least many will go on to chronic pulmonary disease as adults.

II. Complications of Severe Respiratory Disease

1. *Lobar atelectasis:* In about 10 per cent of patients with cystic fibrosis of the pancreas, atelectasis of one or more lobes is present[11] (Fig. 5). In the majority of cases it occurs during the first episode of pulmonary involvement and may lead rapidly to death. In others it accompanies a sudden worsening of long-standing respiratory infection and often represents a terminal complication. In patients who survive, atelectasis generally clears spontaneously within weeks, but at times months after its onset (Fig. 6, A and B). The atelectasis has generally involved the right middle or upper lobes; it has been seen only once in the left lung in the present series.

2. *Mediastinal and subcutaneous emphysema:* In three patients, massive mediastinal and subcutaneous emphysema developed spontaneously (Fig. 7). In all three it was an expression of a sudden exacerbation of the disease, but in only one did it lead to death. In the other two, admin-

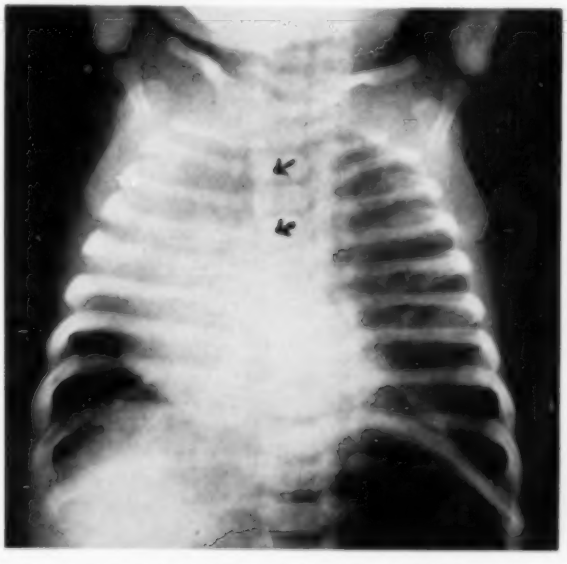


FIGURE 5—T. D., male, age 3 months: Atelectasis of right upper and middle lobes.

istration of antibiotics promptly initiated, both intramuscularly and by inhalation, checked the progress of the pulmonary infection.

3. *Cor pulmonale:* The pressure in the pulmonary vascular bed, high as a result of the chronic pulmonary disease, is markedly and suddenly increased during the repeated attacks of bronchial obstruction which occur during exacerbations of the respiratory infection. This sequence of events at times leads to a rapid dilatation of the heart, visible on x-ray films, and to the clinical signs of circulatory embarrassment.[15] If the patient survives, the heart rapidly returns to its normal size and function, an expression undoubtedly of the intact myocardium in this young age group. Recently in a few patients with long-standing, severe progressive lung disease, there has been found a persistent dilatation of the pulmonary artery on x-ray film (Fig. 8). Polycythemia has generally not been present.

4. *Sudden death from asphyxia:* Sudden death due to coughing up of large amounts of thick, tenacious bronchial secretions which occlude completely the naso-pharynx and cause asphyxia has been seen on many occasions. Indeed it is not an uncommon mode of death in patients debilitated by a chronic wasting illness and unable to cope effectively with an added mechanical insult.

5. *Role of measles and pertussis:* It is not uncommon for the chronic bronchopneumonia in patients with cystic fibrosis of the pancreas to be initiated by measles or whooping cough. This is especially interesting in view of the time-honored observation that rubeola and pertussis are the initial insult leading in some patients to a chronic bronchopneumonia.

Whooping cough is less of a problem at the present time, because of the almost universal prophylactic inoculation of children against this disease. Rubeola, however, frequently leads to severe and at times fatal aggravation of a pre-existing respiratory infection in patients with fibro-

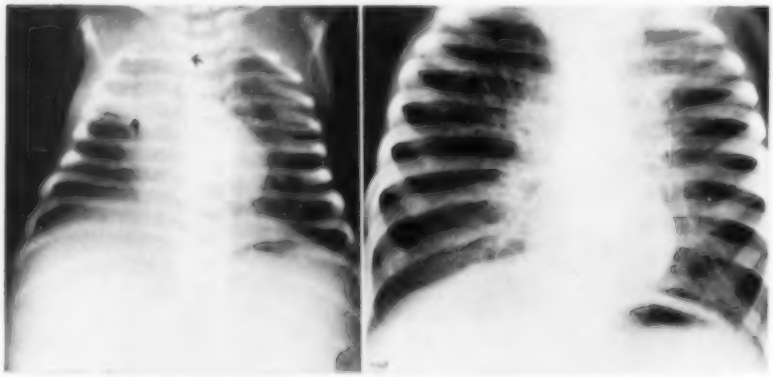


FIGURE 6A FIGURE 6B

Figure 6A: A. H., age two months—Atelectasis of right upper lobe.—*Figure 6B:* age 14 months—Clearing of right upper lobe after atelectasis had persisted unchanged for nine months.

cystic disease. Following subsidence of the fever and disappearance of the rash, bronchial complications may set in.

Patients with cystic fibrosis of the pancreas usually weather without complications the other common contagious diseases of childhood.

III. Diagnosis

In the average case the combination of symptoms of pancreatic insufficiency and of chronic pulmonary disease leads to the clinical suspicion of cystic fibrosis of the pancreas. Definitive diagnosis usually rests on the demonstration of absence of pancreatic ferments on duodenal drainage.[16] Lung tuberculosis can usually be reliably ruled out in children by a negative intradermal tuberculin test.

There are a few patients, however, in whom the lung symptoms predominate and in whom pancreatic activity is not entirely absent on duodenal assay.[17, 18] In such patients the "sweat test" has proved of great diagnostic assistance as it has been shown that the electrolyte content of sweat is increased in fibrocystic disease of the pancreas above that of almost any other disorder.[5, 6] In particular, other conditions characterized by chronic pulmonary disease have a normal sweat chloride and sodium.

The diagnosis of cystic fibrosis of the pancreas, therefore, should be based on the following criteria (Table II): 1) *Pancreatic insufficiency* which leads to steatorrhea, large, bulky, foul stools and the absence of pancreatic enzyme activity on duodenal assay. 2) *Pulmonary pathology*—characterized by obstructive emphysema and chronic bronchopneumonia. 3) *Abnormal sweat* with abnormally increased sweat chloride and sodium and leading to acute salt depletion and at times death in hot weather. 4) *Family history*—Siblings with the same condition. This last point is not absolute as there may be no siblings or there may be several other children who do not have fibrocystic disease. Their presence is therefore of diagnostic assistance, but their absence does not rule against the possibility.

A survey of Babies Hospital records shows that in the last 15 years, chronic bronchopneumonia was entered among the discharge diagnoses in

TABLE II
DIAGNOSIS OF FIBROCYSTIC DISEASE OF PANCREAS

Criteria for Diagnosis	Clinical Manifestations	Test Indicated	Result of Test
1. PANCREATIC INSUFFICIENCY	Steatorrhea Malnutrition	Duodenal Drainage	Absent Pancreatic Enzymes
2. PULMONARY PATHOLOGY	Obstructive Emphysema Chronic Broncho-pneumonia	X-ray film of Chest	——
3. ABNORMAL SWEAT	Salt Depletion in Hot Weather	Sweat Test	Increased Sweat Electrolytes
4. FAMILY HISTORY	Siblings with Cystic Fibrosis of Pancreas	——	——

244 children in addition to their basic disease. Three out of four of these 244 patients had cystic fibrosis of the pancreas, which accounted for the overwhelming number of cases of chronic lung disease in children. Pulmonary tuberculosis was excluded in all instances by repeated intradermal tuberculin tests.

In a previous study it was shown that fibrocystic disease was the leading cause of lobar atelectasis in infants under one year of age.[11]

IV. Treatment of the Respiratory Involvement

The treatment of patients with cystic fibrosis of the pancreas is directed to the pancreatic deficiency and the respiratory involvement.

The effects of pancreatic deficiency are well controlled by an appropriate diet,[19] with a high protein, low fat content, adequate amounts of the liposoluble vitamins and added pancreatic extracts. Because these patients are prone to massive salt depletion through the sweat,[5, 6] liberal amounts of salt should be given in hot weather.

Antibiotic treatment is required by patients who have respiratory involvement.[14, 20, 21] As the basic infection is due to Staphylococcus aureus hemolyticus,[14] penicillin both by intramuscular injection and by inhalation is given, the latter to be considered a form of topical application. One of the wide-spectrum antibiotics—terramycin, aureomycin, achromycin or tetracyn—is added by mouth. If the cultures reveal the presence of gram-negative bacteria, streptomycin both by intramuscular injection and by inhalation is added. Intensive treatment in the hospital is usually given for a period of not less than seven days and not more than 15 days in a single course. Detailed dosages and routes of administration are listed

FIGURE 7 FIGURE 8

Figure 7—E. F., male, age five years: Atelectasis of right lung with shift of trachea and heart. Marked tissue emphysema overlying right hemithorax and neck.—*Figure 8*—A. C., male, age seven years: Enlargement of heart and pulmonary artery shadows after years of severe chronic pulmonary disease. The patient had a right upper lobectomy for persistent atelectasis 3 years previously. The operation failed to influence progress of the disease.

TABLE III
TREATMENT OF RESPIRATORY INVOLVEMENT
IN CYSTIC FIBROSIS OF PANCREAS*

AGENT	ROUTE	DOSAGE
A. THERAPEUTIC COURSE: 7-15 days		
1. BROAD-SPECTRUM ANTIBIOTIC (terramycin, aureomycin, achromycin, or tetracyn)	orally	50 mgm. per Kg. body weight per 24 hours
2. PENICILLIN (procaine)	intra-muscular	400,000 units twice a day
3. STREPTOMYCIN	intra-muscular	1 Gram per 24 hours or 40 mgm. per Kg. body weight
4. PENICILLIN	inhalation**	100,000 units five times a day
5. STREPTOMYCIN	inhalation**	200 mgm. five times a day
B. PROPHYLACTIC ADMINISTRATION: as long as there is respiratory involvement		
1. BROAD SPECTRUM ANTIBIOTIC (terramycin, aureomycin, achromycin or tetracyn)	orally	10-20 mgm. per Kg. body weight per 24 hours

*All dosages for children.

**Penicillin and Streptomycin to be added together to 2 cc. of saline and nebulized (Vaponephrin nebulizer) with oxygen or compressed air flowing at 5 to 6 liters per minute.

in Table III. In recent times erythromycin (40-50 mgm. per Kg. of body weight per 24 hours) in association with gantrisin (0.1 gms. per Kg./24 hours) has been tried with some success.

If patients show evidence of respiratory involvement, continued administration of one of the broad-spectrum antibiotics in prophylactic doses is advisable. This may have to be continued for months or years and it is only when all clinical and radiological signs of pulmonary infection disappear that the drugs can be discontinued.

The problem of resistance of bacteria to antibiotic agents has caused increasing concern in recent years. However, continued administration of these drugs is imperative for the welfare of most patients.

Therapeutic bronchoscopy is contra-indicated because of its difficulty in children, the adverse reactions by which it is often followed and the lack of improvement following the procedure. Lobectomy or partial pneumonectomy for bronchiectasis is clearly not indicated in a patient in whom a diagnosis of cystic fibrosis of the pancreas has been made, because of

the generalized nature of the pulmonary involvement. Lobectomy for persistent atelectasis should be considered if lobar collapse has persisted unaltered for more than one year. Spontaneous expansion of a collapsed lobe may appear even after months (Fig. 6). It should be kept in mind, furthermore, that lobectomy performed for this purpose has generally failed to modify the progressively down-hill course of the disease (Fig. 8). Postural drainage alone is usually ineffective.

In view of the frequency with which measles is followed by serious complications in fibrocystic patients, every effort should be made to prevent or modify this disease by the administration of gamma globulin if exposure is known. If the patient does come down with rubeola, antibiotic treatment is indicated prophylactically.

Immunity against pertussis should be kept up by regular booster injections of the appropriate vaccine.

Finally, administration of digitalis for brief periods may be of assistance in helping the patient overcome transient circulatory embarrassment secondary to an exacerbation of the chronic pulmonary infection.

V. Prognosis

Ten per cent of patients with fibrocystic disease of the pancreas present at birth congenital intestinal obstruction, so-called meconium ileus. About half of these succumb immediately and the other half is operated on successfully. The prognosis thereafter is that of all other children with this condition.

Pancreatic insufficiency with its consequent malnutrition and abnormal stools can be adequately controlled by appropriate dietary measures. Although patients have succumbed not infrequently in the past to acute salt loss in hot weather, this should not happen any longer now that the mechanism of this depletion through the sweat is clearly understood and the physiological needs for sodium and chloride balance in these patients are better known.

The variable degree of bronchial and pulmonary involvement, therefore, determines the fate of the patient. If untreated, the great majority of cases succumb to a progressive chronic bronchopneumonia leading through bronchiectasis and pulmonary insufficiency to death. The average survival subsequent to appearance of the respiratory disease has been two or three years, although any one of the exacerbations of the infection may lead to sudden and fatal increase in bronchial obstruction. The advent of effective antibacterial agents—first penicillin,[14] then aureomycin and terramycin[20, 21] and later other wide-spectrum antibiotics—has greatly improved the outlook for these patients. There are increasing numbers of fibrocystics who with the help of antibiotics weather months and years of relatively mild bronchial and pulmonary infection without permanent damage to their respiratory passages (Table I). These children seem to improve after the age of 10 years, although there are not many who have been able to discontinue entirely the prophylactic administration of antibiotics. This improvement at a certain age may well be due to a purely

mechanical reason, the adverse and dangerous effects of bronchial obstruction being counteracted by the increase in size of these structures. It is not possible to predict the future of these youngsters (the oldest one followed in the present group is 18 years of age), but it would seem logical to assume that at least in some instances chronic lung disease will be present in adult life.

While a varying degree of severity in the involvement of various organ systems is inherent in this disease, there is little evidence that this variability can be influenced by extrinsic factors. Thus controlling the pancreatic deficiency by dietary measures does not seem to influence greatly the pulmonary involvement, except insomuch as good nutrition increases a patient's ability to cope with infection. Severe and at times fatal lung involvement has often developed in patients in whom the disease has been recognized and who had been kept on an appropriate diet for months or years. Conversely, there are a few patients in whom the respiratory involvement has remained minimal despite the fact that complete pancreatic insufficiency had been present, unrecognized, for years. Another important argument has come to light in recent times with the recognition[6] of children and probably adults in the families of patients with cystic fibrosis of the pancreas, who present the pulmonary manifestations and the abnormal sweat characteristic of this disease in the absence of pancreatic involvement.

In conclusion, while prognosis of a patient with fibrocystic disease of the pancreas is serious and at best guarded, it is not hopeless. Of the 292 children with this condition seen to date at Babies Hospital, 133 have died and 159 are living. Of the latter, 29 are from 10 to 18 years of age, some of them doing well.[22] Early diagnosis and administration of antibiotic therapy before the respiratory disease causes permanent damage to the bronchi, in addition to appropriate dietary measures, offers the best hope of tiding these patients over into late childhood when the manifestations of this disorder seem to become milder.

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SUMMARY

Cystic fibrosis of the pancreas, despite its name, is a generalized hereditary disease of children in which the pancreas, the lungs, the sweat glands, and frequently the liver are affected. The basic nature of this widespread disturbances is not known, but it is probable that all exocrine glands are affected in this condition.

A variable degree of bronchial and pulmonary involvement is a virtually constant feature of the disease, usually dominates the clinical picture and determines the fate of the patient. Cystic fibrosis of the pancreas is the commonest cause of chronic non-tuberculous lung disease in the pediatric age group.

Cystic fibrosis of the pancreas differs from other diseases of the lungs

in that the basic phenomenon leading to respiratory involvement is a mechanical one: Failure to remove bronchial mucus, (perhaps abnormal in its physico-chemical characteristics), which results in widespread obstruction and predisposes the patients to secondary infection. Bronchial obstruction may thus be looked upon as the primary and cardinal manifestation of the pulmonary component of fibrcystic disease of the pancreas. The infecting organism is usually Staphylococcus aureus hemolyticus.

Early diagnosis and administration of antibiotics before the respiratory infection causes permanent damage to the bronchi, in addition to appropriate dietary measures, offer the best hope for such patients.

RESUMEN

La fibrosis quística del páncreas a pesar de su nombre, es una enfermedad generalizada hereditaria de los niños en los que el páncreas, los pulmones, las glándulas sudoríparas y a frecuentemente el hígado, está afectado. La naturaleza basida de este transtorno extenso no se conoce, pero es probable que esta condición afecte a todas las glándulas exócrinas.

Una característica virtualmente constante de esta enfermedad es un grado variable de compromiso bronquial y pulmonar, que habitualmente domina el cuadro clínico y determina el futuro del enfermo. La fibrosis quística del pancreas difiere de otras enfermedades pulmonares en que el fenómeno básico que conduce al daño respiratorio es mecánico: consistente de la falta de expulsión del moco bronquial, (el que probablemente es anormal en sus características físico-químicas) lo que produce una obstrucción diseminada y puede así considerarse como la manifestación primaria y cardinal de del compromiso pulmonar de la enfermedad fibroquística del páncreas. El germen infectante habitual es generalmente el estafilococo aureo hemolítico.

RESUME

La dégénérescence fibro-kystique du pancréas, malgré son nom, est une maladie héréditaire généalisée. Elle frappe les enfants, chez qui on constate l'atteinte du pancréas, des poumons, des glandes sudoripares, et fréquemment du foie. La nature véritable de ces troubles disséminés nous échappe, mais il est probable que toutes les glandes exocrines y participent. Les bronches et les poumons sont atteints d'une façon pratiquement constante, à un degré variable, et cette atteinte domine habituellement le tableau clinique, et tient sous sa dépendance l'évolution.

La dégénérescence fibro-kystique du pancréas diffère des autres affections pulmonaires, car dans ce cas, le phénomène essentiel qui détermine les troubles respiratoires est d'origine mécanique. Il s'agit de l'impossibilité d'évacuer le mucus bronchique dont les composantes physico-chimiques sont peut-être anormales. Le résultat en est une obstruction étendue, qui expose les malades à des infections secondaires. Ainsi l'obstruction bronchique peut être considérée comme la première et principale manifestation de l'atteinte pulmonaire de la dégénérescence fibro-kystique du pancréas. Le microbe qui est à l'origine de l'infection est habituellement un staphylocoque doré hémolytique.

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Planigraphy (Body Section Radiography) in Detecting Tuberculous Pulmonary Cavitation

EDWARD A. FAVIS, M.D., F.C.C.P.*
White Haven, Pennsylvania

For this study all planigrams taken at the White Haven Division of the Jefferson Medical College Hospital in White Haven, Pennsylvania, from July 1948 to May 1953 were reviewed. The White Haven Division is a division of the Department for Diseases of the Chest of the Jefferson Medical College Hospital and has an average monthly census of 147 patients.

Body section radiography or planigraphy is not a new technique. It was done originally in France by Bocage in 1921 and by Portes and Chause in 1922. Prior to 1934 an American technician, Jean Kieffer, had perfected and patented an apparatus which he called "The Laminagraph." Dr. Sherwood Moore collaborated with Kieffer in this pioneer work. In 1937 the Semens planigraph was placed on the market and has had extensive use, although it was designed for upright planigraphy and vertical motion. Since then planigraphy is being used in more and more hospitals and sanatoria throughout the country.

During the period under study 320 planigrams were taken on 204 patients (bilateral planigrams were counted as two). Forty-nine taken on 32 patients were not available for review. Therefore, this report will be based on planigrams taken on 172 patients or 85 per cent of the total number of patients that were planigraphed during this period under study. Since 271 planigrams were taken on this group (172) it represents 85 per cent of the total number of planigrams (320) taken. Each planigram was reviewed and interpreted with special emphasis on identifying cavitation.

Planigraphy is now universally used by most chest specialists. The indications for it as followed in this Hospital will be briefly enumerated. The 320 planigrams were taken because one or more of the following conditions were present:

1) Suspicious area or areas for cavitation had been seen in the conventional posterior-anterior, Bucky, or apical lordotic films.

2) "Clear-cut" cavitation had been noted in the conventional films, but further confirmation was desired as to the location and depth of the cavity.

3) Chest surgery was being contemplated on one side and since there was a history of disease in the contralateral lung this side was planigraphed, regardless of the absence of cavitation on this side on the conventional films.

4) Persistent positive sputa in patients who had undergone chest surgery, especially thoracoplasties, in spite of no roentgen evidence of cavitation on the conventional posterior-anterior films.

*Physician-in-Charge of the White Haven Division of the Jefferson Medical College Hospital, White Haven, Pennsylvania.

5) Patients who were definitely known to have had pulmonary cavitation at one time, and regardless of the fact that no cavitation was noted on the conventional views that were taken routinely before a patient was discharged. In these cases planigrams were taken prior to discharge as an added precaution to minimize the possibility of discharging a patient with an undiagnosed cavity.

6) In uncollapsed lungs when persistent positive sputa were present or in patients having hemoptysis and no significant infiltrative disease could be detected in the conventional films.

7) Re-expansion of the lung or lungs collapsed by pneumothorax and/or pneumoperitoneum was being considered. Before the collapse was abandoned planigrams were taken to be further assured that cavitation was no longer present.

8) Decision had to be made whether a patient should undergo pneumothorax, pneumoperitoneum, resection, or thoracoplasty for known tuberculous cavitation. Planigraphy was an aid in gaining some insight as to the thickness of the cavitary wall. (Whenever the cavity was seen as having a thick wall, resection was the treatment of choice provided all other conditions were suitable.)

9) Conventional films revealed suspected honeycombing or bronchiectatic cavitation. (Planigraphy is most helpful in distinguishing between areas of honey-combing and frank cavitation.) This study revealed that by planigrams it is easier to decide whether bronchograms are required to aid in the diagnosis of bronchiectasis.

It is obvious that after careful consideration of all of the above-mentioned indications for planigraphy and after a thorough study of all of the planigrams taken that one would expect to have arrived at a large number of conclusions. However, this discussion is being limited to the role these planigrams played in detecting tuberculous cavitation. Any other knowledge that may have been acquired in reviewing these films will not be included.

All patients in this study have pulmonary tuberculosis and cavities found were considered tuberculous. Planigrams were taken in the recumbent position, and in most instances 4 centimeter to 12 centimeter cuts inclusive were the routine. In certain special cases where cavitation was believed to be present more posteriorly, a 3 centimeter cut was also taken; and when cavitation was believed to be present more anteriorly, 13 and 14 centimeter cuts were included. The group includes 96 planigrams taken of the left lung and 103 of the right lung with 36 taken of both lung fields.

In all instances data were collected on the X-ray findings on the conventional films (posterior-anterior, Bucky, or apical lordotic views) before the planigrams were taken. By this means it was possible to draw conclusions as to the value of planigrams in the light of the findings beforehand on the conventional films. These conventional films were usually taken on the same day as the planigrams, or else earlier, but never earlier than three months.

TABLE I

	Number of Planigrams	Per Cent	
1. No cavitation on conventional view but cavitation on planigrams	29	10.7	53 or 19.5 per cent
2. Suspicious cavitation on conventional view but cavitation on planigrams	24	8.8	
3. No cavitation on conventional view and no cavitation on planigrams	82	30.3	
4. Suspicious cavitation on conventional view but no cavitation on planigrams	51	18.8	
5. Cavitation on conventional view and cavitation on planigrams	37	13.6	
6. Suspicious cavitation on conventional view and suspicious cavitation on planigrams	20	7.4	
7. No cavitation on conventional view but suspicious cavitation on planigrams	11	4.1	
8. Cavitation on conventional view but no cavitation on planigrams	9	3.3	
9. No conventional view available for comparison	7	2.6	
10. Cavitation on conventional view but suspicious cavitation on planigrams	1	0.4	

Of the 271 planigrams included in this study (Table I) in 29 or 10.7 per cent although cavitation was conclusively shown no cavitation was apparent on the conventional films. In 24 or 8.8 per cent the planigrams conclusively showed cavitation, while on the conventional films only suspicious areas were seen. This whole group totals 53 or 19.5 per cent. It is believed that this figure of 19.5 per cent is quite high and certainly justifies the use of planigraphy in discovering tuberculous cavitation. When one considers that planigraphy is not only used to detect cavitation but that other information, can also be acquired, it is felt that its more extensive use should be encouraged.

This study also revealed that in 37 planigrams where cavitation was found it had also been seen on conventional views. In nine planigrams no cavitation could be found, although it was reported as being present in conventional films. In 51 no cavitation was noted, while the posterior-anterior film showed suspicious areas. The planigrams were of no help in 20 cases where the areas in question were suspicious before planigraphy and were still reported as suspicious on the planigrams. In 82 no cavities were seen, by either technique. Eleven planigrams showed suspicious areas for cavitation, while the conventional films showed no cavitation. In one case a cavity was seen on the conventional film and planigrams only revealed a suspicious area. One group of seven planigrams were not included in any of the above groups because no conventional films were available for comparison.

Planigrams should by no means be considered the "last word" in the detection of cavities, so much so that if one received a negative report that it is not to be interpreted as absolute proof that a cavity is not present. This would be most unfortunate because patients might then be treated as if the disease were completely arrested, when in reality a cavity was

present, denoting active disease. In fact, one needs only to review the results cited in the paragraph above to see that conflicting findings were present between conventional posterior-anterior views and planigrams. The detection of pulmonary cavitation in tuberculosis is most important as in one way or another the treatment of this disease is centered on this finding. The present belief held by most specialists is that a cavity denotes the presence of active disease. This is especially true since chemotherapy has rendered the sputa of so many patients negative for tubercle bacilli, while X-ray films still demonstrate disease, particularly pulmonary cavitation. It is felt that the presence of a cavity, is the surest sign of failure of or inadequate treatment of this disease.

Planigraphy should be considered one of those procedures to help in detecting cavitation, without which the examination is incomplete. It is further felt that knowledge gained from planigrams should be used with any other findings that may be present, but hardly ever as the sole means of disposing of a case.

When planigrams show "clear-cut" cavitation the interpretation is relatively simple. Unfortunately most planigrams do not reveal cavities so clearly.

It has been found that films most difficult to interpret are planigrams taken of portions of lungs collapsed by thoracoplasty, since not all radiolucencies seen are cavities. The following rule has been most helpful: "Before any radiolucency can be called a cavity its walls can be made up of no such structures or part of structures as blood vessels, ribs, transverse processes, or bodies of vertebrae, fibrotic strands, or thickened pleura, walls of a bronchus, or any of the spicules of regenerated rib so often seen in patients who have had thoracoplasties."

At times even when this rule is followed, it has been impossible to definitely confirm or deny the presence of a tuberculous cavity. Cases such as these require repeat planigrams. It is often easier to over-read than under-read planigrams. For example, one set may be interpreted as showing presence of cavity, while subsequent ones may reveal that the pre-

TABLE II

	Total	*Patients*	*Per Cent of Planigrams Taken*
Planigrams taken from July 1948 to May 1953	320	204	
Planigrams in study	271	172	85
Planigrams unavailable (omitted from study)	49	32	15

TABLE III

	Number of Planigrams	*Per Cent*
Right Planigrams	103	44
Left Planigrams	96	41
Bilateral Planigrams	36	15

viously reported radiolucency was made up only of fibrotic strands or other structures and not a cavity.

Honey-combed areas seen in such a large number (37 or 13.6 per cent) of the total planigrams reviewed, were not labelled cavities and thus were not included in the group demonstrating definite cavitation on planigraphy. Honey-combed areas were practically all noted in the upper lobes with single or multiple small radiolucencies usually one-half centimeter or less in diameter. These "punched-out" or "moth eaten" areas are referred to by others as "bronchiectatic cavities." Suffice it to say that many patients with planigrams of this type had bronchograms performed and bronchiectasis was found. From the knowledge that was acquired from these planigrams, it is felt that they are not only important in detecting pulmonary tuberculous cavities but are also useful in suspecting or even diagnosing bronchiectasis. Therefore, from the observations noted on these planigrams one is justified in requesting bronchograms for a definite diagnosis.

SUMMARY

A review of 271 planigrams taken over a period of four years and 11 months at the White Haven Division of the Jefferson Medical College Hospital revealed that 19.5 per cent conclusively demonstrated cavitation, while on the conventional films either no cavity or only a suspicious area was noted. This figure is considered significantly high to justify the more liberal use of planigraphy in the detection of tuberculous pulmonary cavitation.

The indications for planigraphy were briefly enumerated. However, the discussion was limited to the role of planigraphy in detecting tuberculous cavitation.

One should not rely entirely and completely on planigrams in detecting cavitation, because there will be cases in which cavitation is missed. In cases of doubt frequent check-ups, including repeat planigraphy, are indicated.

How one would be able to minimize the possibility of calling all radiolucencies seen on planigrams cavities was discussed.

This study demonstrated the value of planigraphy as an aid in the differentiation between small tuberculous cavitation and "bronchiectatic cavitation" and further helped in deciding which patients required bronchograms to diagnose bronchiectasis.

RESUMEN

La revisión de 271 planigrafías tomadas en cuatro años y 11 meses en la Sección White Haven del Hospital de la Escuela de Medicina Jefferson, reveló que 19.5 por ciento demostraron concluyentemente la existencia de cavernas en tanto que en la película corriente no había cavidades o sólo se sospechaban. Esta cifra se considera significativamente elevada para justificar el uso más liberal de la planigrafía en el descubrimiento de las cavernas tuberculosas.

Se indicaron brevemente nuestras indicaciones para la planigrafía. Sin

embargo, la discusión se ha limitado al papel de la planigrafía para descubrir las excavaciones tuberculosas.

No debe uno confiar completamente en los planigramas para descubrir las cavidades porque hay casos en que las excavaciones se escapan. En caso de duda, están indicadas las frecuentes revisiones incluyendo repetidas planigrafías.

Se discute como podría uno disminuir las posibildades de creerque todas las áreas claras vistas en las planigrafías sean cavidades.

Este estudio demostró el valor de la planigrafía como auxiliar en el diferenciación entre pequeña caverna tuberculosa y "cavidad bronquiectásica" y más aún, para decidir qué enfermos necesitan broncogramas para diagnosticar la bronquiectasia.

RESUME

L'auteur fait l'étude de 271 tomographies faites pendant une période de quatre ans et onze mois à la Division de Port-Blanc de l'Hôpitau Universitaire Jefferson. Il a constaté que dans 19,5% des cas, on avait pu mettre en évidence d'une facon certaine une cavité alors que sur les films standard, on ne constatait pas de caverne ou on constatait simplement une zone suspecte. Ce pourcentage peut être tenu comme suffisamment élevé pour justifier une utilisation plus large de la tomographies pour la recherche des cavernes tuberculeuses du poumon.

L'auteur énumère rapidement ses indications de tomographies. Toutefoies, il limite le problème dans ce cas au rôle de la tomographie dans la découverte des cavernes tuberculeuses.

Il ne faut pas se fier entièrement et intégralement sur lestomographies pour découvrir les cavernes, car dans certains cas, elles peuvent ne pas être mises en évidence. Il faut faire de fréquents examens complets en répétant la prise de tomographies.

Cette étude a démontré la valeur de la tomographie dans le diagnostic différentiel entres les petites cavernes tuberculeuses et les bronchiectasies kystiques. Elle a permis de décider des cas pour lesquels il fallait compléter les investigations par la bronchographie pour permettre le diagnostic de dilatation bronchique.

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Ineffectiveness of Isoniazid as an Appetite-Stimulator*

A. GERSON HOLLANDER, M.D., F.C.C.P.
Oakland, California

In 1952 the clinical use of isoniazid in the treatment of pulmonary tuberculosis was described by Robitzek and Selikoff,[1] and by McDermott and his associates.[2] Although both groups of workers were primarily interested in the therapeutic effects of the drug on the clinical course of the disease, one of the outstanding subjective responses of the patient was the marked increase of appetite. As Robitzek and Selikoff[1] put it: "The most descriptive stock expression is 'ravenous.'" Weight gains ranging up to 59 pounds were recorded over a period of four to 15 weeks, and the average weight gain was 19.7 pounds in 8.8 weeks of therapy. They noted that the weight gains usually became evident in the second or third weeks. It seemed possible that the appetite-stimulating effect of the drug might not be entirely dependent on the reduction in toxicity of the tuberculous process, and it was felt that this possibility should be investigated.

Plan of Investigation

The 20 patients chosen for study were all adult males without evidence of pulmonary tuberculosis or tuberculous infection. They were long-term patients who were being treated on the Neurological and Orthopedic Services for a variety of disorders such as residuals of poliomyelitis, hemiplegia, paraplegia, fracture dislocation of the cervical vertebrae, multiple sclerosis, etc. Tuberculosis had been carefully excluded on the basis of history, complete physical examination and x-ray film of the chest. Blood urea nitrogens were obtained prior to starting the drugs and were within normal limits. Weights were obtained at weekly intervals, and the weight curve was constant for three weeks prior to testing. All selected had either "poor" or "fair" appetites, confirmed by the physician and nurse. The only information given to the patients was that they were being given a vitamin tablet. The same nurse made a notation as to the status of their appetite prior to the initiation of the experiment, and weekly thereafter. Objectively, a note was made as to whether the patient consumed an increasing quantity of food. One tablet was given three times a day for a period of 14 days. A rest period of 10 to 14 days was allowed before the patient was submitted to retesting with another preparation. Twenty were placed on each of the preparations for a period of two weeks. Weight and appetite changes were recorded weekly. In no instance were untoward effects noted except for flushing when preparation "M" was used.

Chief, Tuberculosis Service, Veterans Administration Hospital, Oakland, California.
*From the Veterans Administration Hospital, Oakland, California.

Preparations: Four tablets were used, all identical in size, shape and color, but consisting of (1) 100 mg. of isonicotinic acid hydrazid, (2) 100 mg. of the isopropyl derivative of isonicotinic acid hydrazid, (3) 50 mg. of nicotinic acid, (4) lactose. These preparations were labeled "K," "L," "M," and "N," the code being kept under seal so that neither the staff nor the investigator was aware of the identity of the preparation being given.

Results

The results (table) were uniform in that no significant weight change was noted in any of the groups nor was there any change in appetite. One preparation ("M") had to be discontinued because of the flushing reaction which was obtained immediately after its use. It became apparent that this was nicotinic acid, and the probabilities are it would have been wiser to have used a 25 mg. dose.

Drugs	*No. Pts.*	*Pre-Test Appetite* *Fair*	*Poor*	*Change in Appetite*
"K" (Lactose)	20	12	8	0
"L" (Isoniazid)	20	14	6	0
*"M" (Nicotinic Acid)	20	17	3	
"N" (Iproniazid)	20	15	5	0

*Reactions occurred and necessitated discontinuing drug before completing experiment.

ACKNOWLEDGEMENTS

The author is indebted to Dr. H. Corwin Hinshaw for suggestions in conducting this study, to Dr. J. K. Smith, Dr. C. S. Ushiro and the nurses of the Neurological Service for their aid in the study, and to Dr. Lawrence B. Hobson and the E. R. Squibb and Sons Company who made these preparations available.

CONCLUSIONS

In 20 patients who were not suffering from tuberculosis and whose weight had been constant for a period of weeks, but whose appetites were "poor" or "fair," no appetite-stimulating properties were demonstrated by the administration of 300 mg. of isonicotinic acid hydrazid daily, or 300 mg. of the isopropyl derivative of isonicotinic acid hydrazide daily. It is therefore probable that the effect on appetite and weight gain as reported by Robitzek and Selikoff,[1] as well as McDermott and others,[2] was secondary to the suppression of toxicity and not an independent effect of the isonicotinic acid hydrazide derivatives on the appetite.

RESUMEN

Se trató de encontrar si era posible obtener un efecto estimulante del apetito en 20 enfermos que no padecían tuberculosis, dando en estos enfermos que tenían un peso constante, 300 miligrams de hidracida del ácido isonicotínico o de su derivado isopropílico en la misma dosis diariamente y no se comprobó propiedad estimulante del apetito.

Por tanto, es probable que el efecto sobre el apetito y sobre elpeso tal como lo relatan Robitzek y Selikoff[1] así como por McDermott y otros[2] sea secundario a la supresión de la toxicidad y no un efecto independiente de la isoniacida y derivados sobre el apetito.

RESUME

Les auteurs ont étudié 20 malades non tuberculeux, dont le poids était resté constant depuis plusieurs semaines, mais qui avaient un mauvais appétit. On leur administra 300 mmg. par jour d'hydrazide d'acide isonicotinique ou 300 mmg. de son dérivé isopropyle. L'appétit ne fut nullement stimulé par ce traitement. L'explication probable en est que le gain d'appétit et de poids dû à l'isoniazide (et tel qu'il a éte rapporté par Robitzek et Selikoff, ainsi que par McDermott et ses collaborateurs) est secondaire a la suppression de la toxicité de la maladie et non pas consécutif à l'effet particulier de dérivés de l'acide isonicotinique sur l'appétit.

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Lipoid Pneumonia
Due to Prolonged Ingestion of Mineral Oil

JOHN B. MEYERS, M.D., St. Louis, Missouri, and
ROBERT L. GRIFFITH, M.D., Seattle, Washington

Aspiration of certain oils into the bronchial tree is known to cause an inflammatory reaction.

Whereas the earlier reports called attention to this condition in children following the use of oily nosedrops or aspiration of cod liver oil, cream or other fats, in recent years there has been an increasing number of reports of cases among adults. A review of the literature in 1936 revealed 106 cases of which 39 were in adults. Freiman, et al.,[1] in 1940 brought the total number of cases in adults to 105. Sweeney[2] in 1943 collected 264 cases in adults. Volk, et al.,[3] in 1951 found lipoid pneumonia in 14.6 per cent of a group of 389 chronically ill patients in one institution.

Various oils cause different reactions in the lung.[4] Vegetable oils such as poppy seed and sesame oil, which are commonly used in bronchography, produce little reaction in the lung. Fish and animals oils produce an acute hemorrhagic or necrotizing reaction. Mineral oil produces a mild foreign body type of reaction. Mineral oil does not stimulate the cough reflex when it enters the tracheobronchial passages. The pulmonary reaction varies according to the amount of oil and the duration of the process. The dependent portions of the lungs are most commonly involved; that is, the bases in ambulatory patients and the posterior portions in bed patients. The right side is more often involved than the left. The process frequently simulates bronchopneumonia. On gross examination the involved area may be a yellow or brown color and oil may be expressed by pressure or scraping with a knife. On microscopic examination the alveolar walls and interstitial tissues show edema and cellular infiltration including large mononuclear cells containing oil droplets. Later the appearance is that of chronic interstitial pneumonia. Following this there is pulmonary fibrosis and possibly atelectasis and bronchiectasis. There may be circumscribed lesions, sometimes called paraffinomas, which may resemble neoplasms, cysts or abscesses.

The roentgenographic changes are not specific for this condition and are usually described as bronchopneumonia, hilar clouding, increased bronchial markings, lobar pneumonia or tumor.

Of 355 autopsies performed at this hospital from July 1, 1949, to July 1, 1952, the following seven cases showed lipoid pneumonia as a complicating or concomitant disease.

Case 1: C. M., a 60 year old white male, was first admitted to the hospital September 28, 1950, with hypertensive cardiovascular disease of several years' duration. He also gave a history of duodenal ulcer for 12 years. In his systemic history he stated that he had been taking mineral oil for constipation for a number of years. On examination the blood pressure recordings ranged from 259 mm. Hg. systolic and 180 mm. Hg. diastolic to 210 mm. Hg. systolic and 160 mm. Hg. diastolic. Roentgenogram of the chest showed enlargement of the heart and aorta and prominent lung markings which were interpreted as evidence of congestion. In addition to digitalis and other

U. S. Public Health Service Hospital, Seattle, Washington.

supportive therapy, he was given mineral oil on several occasions. On March 10, 1951, he was admitted for the third time complaining of headaches and precordial pain. Temperature was 37.8 degrees Centigrade. Pulse was 82. There was a systolic murmur at the base of the heart. Rales were present in both lung bases. The roentgenogram of the chest (Fig. 1) showed extensive pneumonic infiltration in both lower lung fields and congestion throughout the chest. The electrocardiogram showed left ventricular hypertrophy and strain. He became more dyspneic and cyanotic and died the day after admission.

On necropsy the heart was enlarged and the aorta was enlarged and tortuous. On histologic examination there were multiple small thromboses of the cerebral and coronary arteries. On gross examination of the lungs the posterior portions were firm and the cut surfaces were mottled and of a pale, yellow-tan color. Histologic Examination (Fig. 2) showed the parenchymal pattern to be distorted by the presence of granulomatous tissue. The alveoli were almost completely filled with macrophages containing many large and small vacuoles and macrophages containing brownish pigment. The alveolar walls showed fibrous thickening. Vascular engorgement was not prominent and almost no neutrophils were present. Sudan fat stain showed sudanophilic material within the macrophages.

Case 2: J. F. G., a 47 year old white male, was first admitted to the hospital October 4, 1944, with left hemiparesis. At that time the blood pressure was 184 mm. Hg. systolic and 120 mm. Hg. diastolic. Laboratory examinations of the blood, urine and cerebrospinal fluid revealed no abnormalities. He was next admitted in October 1947 with multiple burns when he stated that he suffered from chronic constipation for which he had been taking mineral oil regularly for about 11 years. He was admitted for the last time April 21, 1951, in a state of mental confusion. His temperature was 36.8 degrees Centigrade, pulse 92 per minute and respirations 18 per minute. There was atrophy of the muscles of the extremities on the left and also weakness of the muscles on the right. The heart sounds were normal. There were rales at both lung bases. He died the day following admission.

Necropsy revealed evidence of several acute and subacute myocardial infarctions and cerebral arteriosclerosis. The posterior one-half of each lower lobe showed congestion and multiple yellow-gray indurated areas measuring up to 3 cm. in diameter. These were sharply defined but not encapsulated. There was a suggestion of oiliness of the material expressed from these areas. On histologic examination (Fig. 3) these indurated areas showed a granulomatous, proliferating, inflammatory reaction completely destroying the normal alveolar architecture and poorly demarcated from the surrounding lung tissue. Included in these areas were large numbers of macrophages containing vacuoles from which lipoid material had been dissolved, multinucleated macrophages, proliferating fibroblasts and infiltrations of lymphocytes. The apices of both lungs showed emphysema.

FIGURE 1 (CASE 1): Extensive consolidation due to lipoid pneumonia.

Case 3: H. P., a 61 year old white male, was first admitted to the hospital December 13, 1948, with a diagnosis of pituitary tumor of 18 years' duration. He was totally blind and showed the characteristic picture of pituitary deficiency. The roentgenogram of the skull showed an enlarged pituitary fossa and erosion of the clinoid processes. A 70 mm. roentgenogram of the chest was normal. He was readmitted October 14, 1949, in a moribund condition and died 45 minutes later.

Necropsy revealed a chromophobe adenoma of the pituitary, bronchopneumonia and lipoid pneumonia. There was consolidation of the right upper and left lower lobes. Histologic examination showed areas where the alveoli were filled with fat-laden macrophages. Some areas also contained neutrophils, extracellular vacuoles and cells containing brown granular pigment.

Following postmortem examination a history was obtained from the relatives that the patient had been taking mineral oil almost daily for many years because of chronic constipation.

Case 4: A. K. H., a 63 year old white male, was first admitted to the hospital July 1, 1943, with right-sided hemiplegia from a cerebrovascular accident. He was readmitted April 30, 1945, because of a urinary tract infection. Examination revealed a senile, debilitated man with paralysis of the right arm and leg. Blood pressure was 180 mm. Hg. systolic and 110 mm. Hg. diastolic. The prostate gland was greatly enlarged. Urinalysis showed three plus albumin, gross pus, hyaline and granular casts. He recovered from the acute urinary tract infection but remained bedridden until death March 7, 1951. During this time his illness was complicated by arteriosclerotic heart disease with auricular fibrillation and cardiac decompensation, and thrombophlebitis of the right leg. He had pneumonia in October 1950 at which time the roentgenogram of his chest (Fig. 4) showed congestion, increased markings and a density in the left midlung field. At the time of death he had what appeared to be terminal bronchopneumonia.

Necropsy revealed arteriosclerosis of the coronary arteries, myocardial fibrosis, myocardial hypertrophy, calcific valvulitis, encephalomalacia of the left frontal and parietal lobes and of the basal ganglia. The posterior portions of both lungs were consolidated and of uniform light gray color. Purulent, gray, frothy material could be scraped from the cut surfaces of both lungs except for a portion of the left upper lobe. The alveoli contained many cells with vacuolated cytoplasm, many neutrophils and pink amorphous material. The more solid areas showed fibroblastic organization varying from loose to dense fibrosis. In some areas the alveoli appeared like vacuoles in a dense fibrous stroma.

On reviewing his chart it was found that mineral oil had been given to him frequently for constipation during the past six years.

Case 5: C. P., a 74 year old white male, was first admitted May 25, 1946, because of a cerebrovascular accident manifested by aphasia and inability to swallow. There was also a history of attacks of precordial pain and epigastric distress. On examination the blood pressure was 220 mm. Hg. systolic and 124 mm. Hg. diastolic. There was almost complete pharyngeal paralysis. Generalized arteriosclerosis was present. The urine contained albumin. His treatment included intragastric feedings through a nasal tube lubricated with mineral oil.

He was readmitted June 8, 1949, complaining of precordial pain on exertion. He stated that he had had another cerebrovascular accident the previous month and that he still had epigastric distress. He had been constipated and had been taking mineral oil but could not recall how long. He appeared chronically ill. The blood pressure was 190 mm. Hg. systolic and 140 mm. Hg. diastolic. The heart was enlarged and there was a basal systolic murmur. The roentgenogram of the chest was not remarkable. The prostate gland was enlarged. On June 24, 1949, the chest roentgenogram was repeated (Fig. 5) and revealed an area of clouding extending downward from the right hilus. The leukocyte count was 6,600 per cubic millimeter. The erythrocyte sedimentation rate was normal. An electrocardiogram showed evidence of diffuse myocardial disease. Subsequently a barium enema showed numerous diverticula of the descending and sigmoid colon. He remained afebrile until the last three days when the temperature reached a maximum of 38.0 degrees Centigrade. He became gradually weaker and died July 18, 1949.

Necropsy showed generalized arteriosclerosis, coronary arteriosclerosis, and old posterior myocardial infarction, myocardial hypertrophy and fibrosis, cerebral arteriosclerosis and focal encephalomalacia of the pons and thalamus. On gross examination of the lungs the right upper and middle lobes were uniformly dense. The cut surfaces were dull, gray-red and a pale gray exudate was present. The right lower lobe was congested. Histologic examination of the right upper and middle lobes showed the alveoli partly filled with large cells containing many vacuoles in the cytoplasm and some cells containing finely granular, golden pigment. The alveolar walls were widened by proliferation of fibroblasts and of the alveolar endothelial cells. In places there was blending of the fibroblasts in the alveolar walls and in the alveolar spaces. There was moderate vascular congestion.

Case 6: R. J. F., a 50 year old white male, was admitted September 23, 1949, with stricture of the esophagus caused by swallowing lye in early childhood. Since 1931 he had been dilating the stricture with bougies, using mineral oil as a lubricant. Ten months prior to admission he noticed a sensation of a new stricture higher than the old one. This progressed to such an extent that he was unable to pass the bougie himself. He also stated that he had had pneumonia in 1929, 1942 and 1948. The last two attacks followed dilatation. Recently he had been coughing up dark cylindrical masses. He was afebrile but roentgenograms of the chest showed increasing consolidation of the right upper lung field and increased markings in the middle and lower fields (Fig. 6). The leukocyte count was normal. Bronchoscopy showed the right lateral tracheal wall to be pushed in by an extraluminal mass. Esophagoscopic examination showed the anterior margin of the esophagus to be irregular and necrotic beyond the inferior pharyngeal stricture. Biopsy of this area showed anaplastic tumor tissue. Following this gastrostomy was done but he died December 7, 1949.

On necropsy there was carcinoma of the esophagus with metastases to the mediastinum, right upper lung, liver, the thyroid and the adrenal gland. The right upper lobe contained an abscess in the carcinomatous tissue. The right middle and lower lobes showed lipoid pneumonia as evidenced by fibroblastic proliferation and cells containing lipoid material.

A review of his chart showed that he had also used mineral oil regularly as a laxative.

Case 7: N. D., a 30 year old white male, was admitted January 24, 1949, complaining of a painful mass at the left side of his neck of nine months' duration. Examination was not remarkable except for a hard mass 6 cm. by 8 cm. below the angle of the jaw and smaller nodules adjacent to it. Biopsy of this showed what was believed to be an adenocarcinoma. Following this hemithyroidectomy and radical neck dissection were done. He was readmitted September 6, 1949, with generalized metastases and died October 24, 1949, from a massive cerebral hemorrhage. A roentgenogram of the chest on September 15, 1949, showed only increased lung markings.

Necropsy showed disseminated malignant melanoma of undetermined origin. An incidental finding was that of lipoid pneumonia with large cells containing vacuolated cytoplasm and brown granular pigment. There were only two small metastatic nodules in the lungs.

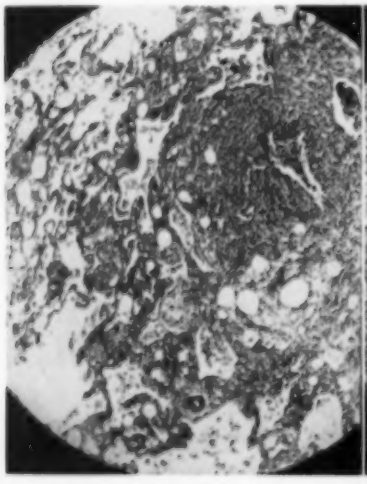

FIGURE 2

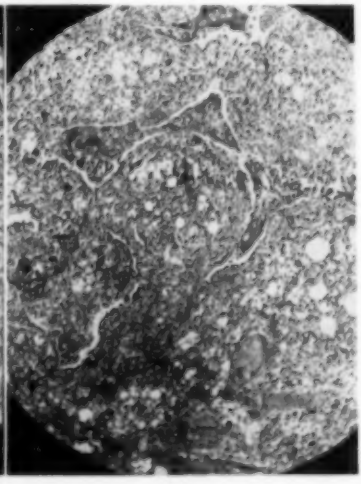

FIGURE 3

Figure 2 (Case 1): Granulomatous reaction, including fibroblasts and macrophages containing vacuoles from which lipoid material has been dissolved. The lipoid material was demonstrated by a Sudan fat stain.—*Figure 3* (Case 2): A granulomatous proliferative inflammatory reaction completely destroying the normal alveolar architecture. Macrophages are seen containing vacuoles from which lipoid material has been dissolved. Multinucleated macrophages and focal infiltrations of lymphocytes are also present.

His wife stated that he had been taking mineral oil about every other day for constipation.

Discussion

Each of the seven patients presented had some serious illness other than lipoid pneumonia which was primarily responsible for death. However, in three cases 1, 4 and 5 the pathologist listed lipoid pneumonia as the immediate cause of death. In case 2 lipoid pneumonia was considered to be an important contributory cause of death. In cases 3, 6 and 7 lipoid pneumonia was present as an incidental but a definite finding. All of the patients had used mineral oil regularly as a laxative. Case 6 had also used it to lubricate the bougie used to dilate his esophageal stricture.

Lipoid pneumonia occurs in all age groups from infancy to senility. Schneider[5] reported several cases of oil pneumonitis in apparently healthy adults and pointed out the difficulty in diagnosis because of the benign early phase of this condition. We have recently treated a man who gave a history of taking mineral oil for over 10 years, with recent development of cough, dyspnea and frequent upper respiratory infections. The roentgenogram of his chest showed prominent markings throughout both lungs and linear density in the right mid-lung field. Lipoid material was demonstrated in his sputum with Sudan III stain. Bronchoscopic examination was not remarkable. Bronchograms showed minimal bronchial dilatation in the right lower lobe. It was felt that he was developing early bronchiectasis associated with lipoid deposits. It is hoped that avoidance of mineral oil will halt or delay the progress of his disease.

In suspected cases of lipoid pneumonia it is advisable to have the patient on a fat free diet for three days before collecting sputum specimens. This helps avoid oral contamination of the specimen. Fresh unstained sputum may reveal oil droplets or, when stained with Sudan stains, the sputum may reveal phagocytic cells containing oil. It has been reported that a bedside test may be made by bringing the sputum in contact with light tissue paper and looking for oil droplets clinging to the paper. Needle aspiration of the lung may reveal oil or oil-containing tissue.[5, 6] Some caution must be exercised in making the diagnosis of lipoid pneumonia from the presence of fat in the sputum. Fat from food particles may contaminate the sputum. It has also been shown[7] that microscopic fat is present in the sputum of normal people and in tuberculous patients. This fat is predominantly imbedded in the mucin.

Awareness of lipoid pneumonia in the differential diagnosis of tumor-like roentgenographic shadows in the chest may prevent an unnecessary thoracotomy. This is especially true at the present time when more routine chest roentgenograms are being taken and when more radical surgical procedures are attempted in undiagnosed pulmonary lesions.

The following is a list of some of the conditions in which mineral oil is frequently used, with or without the advice of a physician:

1. Nasal disorders.
2. Perforated ear drum.
3. Oral disorders which interfere with the swallowing mechanism.
4. Pressure in the throat or neck from tumors, lymphatic enlargement, or goiter.
5. Scarring and contracture from tracheotomy or other causes.
6. Esophageal diverticulum.

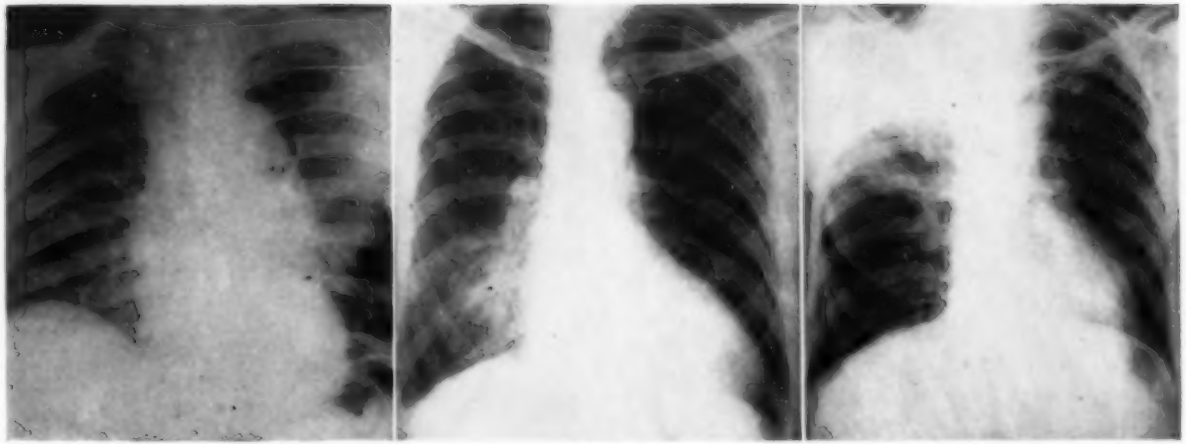

FIGURE 4 FIGURE 5 FIGURE 6

Figure 4 (CASE 4): Roentgenogram, taken while the patient was in bed, shows prominent bronchial markings and density on the left.—*Figure 5* (CASE 5): Area of clouding extending downward from the right hilus.—*Figure 6* (CASE 6): Postmortem examination showed carcinoma in the right upper lobe and lipoid pneumonia in the right middle and lower lobes.

7. Esophageal stricture.
8. The passage of tubes, instruments or dilators lubricated with mineral oil.
9. Cardiospasm with regurgitation and aspiration.
10. Tumor or polyp in any part of the gastrointestinal tract.
11. Esophagitis, esophageal ulceration or esophageal varices.
12. Peptic ulcer with or without pyloric obstruction.
13. Spastic colitis, mucous colitis, atonic colon.
14. Prostatic hypertrophy or prostatitis.
15. Pregnancy, pelvic tumors.
16. Hemorrhoids, anal fissure, fistula or stricture.
17. Constipating diets, dehydration.
18. Neurologic disorders such as cerebrovascular accidents, multiple sclerosis, parkinsonism, especially with loss of gag or cough reflex.
19. Cardiovascular diseases such as myocardial infarction, cardiac decompensation, thrombophlebitis. Frequently physicians advise mineral oil to prevent straining at stool.
20. Any condition associated with prolonged bed rest and inactivity, such as tuberculosis, fractures, senility, psychoses.

The examiner's index of suspicion should be raised when an obscure pulmonary disorder is associated with any of the above conditions and the patient should be questioned specifically regarding the use of mineral oil.

Proudfit, et al.,[8] recently reported a case of chronic lipoid pneumonia following the prolonged inhalation of mineral oil spray used in the man's occupation.

In addition to the pulmonary hazards, it is recognized that excessive use of mineral oil decreases the absorption of carotene and vitamin A. This is true not only in its medicinal use but also in foods and cooking.[9]

SUMMARY

1. Seven cases of lipoid pneumonia found at postmortem examination are presented. These represent 2 per cent of the postmortem examinations at this hospital for a three-year period. All of the cases had other serious illnesses but in at least four, lipoid pneumonia was believed to be an important contributory cause of death. The habitual use of mineral oil was believed to be the cause in all cases.

2. In the case of proliferative process due to a known foreign body, the term chronic pulmonary oleosis seems more appropriate than lipoid pneumonia.

RESUMEN

1. Se presentan siete casos de neumonía lipóidica encontrados en la autopsia. Estos casos representan dos por ciento de los exámenes de este hospital durante tres años. Todos los casos han tenido enfermedades serias, pero por lo menos en cuatro se cree que la neumonía lipóidica, fué un factor importante en la causa de la muerte. El uso habitual del aceite mineral, se cree cue es la causa en todos los casos.

2. En el caso de un proceso proliferativo debido a un cuerpo extraño conocido, el término oleosis crónica pulmonar, parece más adecuado que el neumonía lipóidica.

RESUME

1. Les auteurs rapportent sept cas de pneumonie lipidique découverte à l'autopsie. Cette affection reprèsente 2% de l'ensemble des autopsies pratiquées dans leur hôpital pendant une période de trois ans. Dans tous

les cas, il y avait une autre affection grave surajoutée. Cependant, dans au moins quatre d'entre eux, on pouvait admettre que la pneumonie lipidique avait joué un rôle important dans la mort. Les auteurs pensent que dans tous les cas, la cause en était l'usage habituel d'huile minérale.

2. Dans le cas où il s'agit d'un processus prolifératif dû à un corps étranger connu, il parait plus normal d'utiliser le terme de "pneumonie chronique huileuse" que celui de pneumonie "lipidique."

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Case Report Section

Spontaneous Rupture of the Esophagus

J. A. ARATA, M.D., C. G. McEACHERN, M.D., F.C.C.P., and H. ZWICK, M.D.
Ft. Wayne, Indiana

Spontaneous rupture of the esophagus has attracted relatively little attention from the medical profession, and until recently, it has seldom been mentioned in the differential diagnosis of acute upper abdominal and chest pain.

The first recorded case was that of Boerhaave[1] in 1724. His patient, Baron Wassenaer, Grand Admiral of the Dutch Navy, frequently overindulged in eating and often induced vomiting in the Roman fashion. One day in 1723, after induced vomiting following a heavy meal, he was seized with agonizing chest pain which caused him to feel a bursting sensation in the chest and to collapse and roll about in agony, retching but not vomiting. For 18 hours, he was unrelieved of his suffering despite all efforts of his attending physician. Postmortem examination disclosed gastric content free in both pleural cavities due to rupture of the esophagus, a short distance above the diaphragm.

Until recently, such a catastrophe remained largely an autopsy room curiosity. Prior to 1946, Eliason and Welty[2] reported the mortality to be 100 per cent excluding two cases reported by Graham.

Early in 1946, Barrett[3] made the correct diagnosis, performed thoracotomy, sutured the esophageal defect, and his patient recovered. Later in 1946, Olsen and Clagett[4] were able to make the diagnosis immediately after rupture, and within three hours the defect was repaired transthoracically with success. To these men must be given great credit for their pioneering approach to this problem.

In a review of the literature in 1952, Ware, Schnider, and Davis[5] collected a series of 86 cases that they felt fulfilled the criteria of spontaneous esophageal rupture. They were able to ascertain that 18 of these cases had recovered following surgery.

Definition: Spontaneous rupture of the esophagus refers to a normal esophagus rupturing from an excessive intraluminal pressure, generally induced by vomiting, and excluding those perforated by instrumentation, by ingested foreign bodies, or those due to malignant or benign ulcers. An esophagus that has been strictured by esophagitis should not be included. One that is associated with a hiatal hernia, is likely afflicted with esophagitis. Likewise, the esophagus of an individual who has been retching for days, may well be eroded due to peptic ulceration.

Site of Rupture and Mechanism: Ware, et al[5] found the left side of the esophagus torn in 39 cases, the posterior side in 11, and the right side in eight. The average length of the tear in the recorded cases has been 2.24 cm., with extremes from 0.6 cm. to 8.9 cm.

McKenzie[6] in 1884, Burt[7] in 1931, Hertzog[8] in 1947, and Mackler[9] in 1952 performed distention experiments on segments of the intestinal tract of cadavers. All authors found the stomach less able to withstand intraluminal pressure than the esophagus before rupturing. The esophagus could occasionally tolerate pressures up to seven pounds per square inch although it generally ruptured between three and four pounds pressure per square inch.

Some have denied the possibility of the normal esophagus rupturing from vomiting. They point out that many cases so reported have had repeated vomiting and probable peptic esophagitis. Some reports have included cases with stricture proximal to the rupture; other reports have been of congenitally short esophagus in conjunction with diaphragmatic hernia. A significant number of cases have had associated gastric and duodenal ulcers.

Because of experimental observations on the cadaver that the esophagus withstands pressure better than the stomach, these authors have concluded that spontaneous rupture from vomiting can never occur in an esophagus that has not been the site of weakening from previous disease.[10]

On the other hand, most reported cases have no symptom to suggest previous esophageal disease, and clinically are spontaneous ruptures. Is is possible for humans to generate sufficient pressure to rupture the esophagus? Kinsella[11] states that a well muscled individual can blow pressures of 3.44 to 3.63 pounds per square inch. No one knows just how much pressure is created in vomiting.

A consideration of the mechanism of vomiting discloses how the esophagus can be ruptured and the stomach left intact *in vivo*, even though the stomach in the cadaver is less able to tolerate pressure without bursting, than is the esophagus.

The propulsive forces is supplied by a great increase in intraabdominal pressure. This latter is brought about by contraction of the abdominal wall muscles simultaneously with the diaphragm. At the same time, there is contraction in the region of the incisura and relaxation of the cardia and the esophagus. The intragastric pressure thus is increased largely due to compression from without and the uniform pressure surrounding the stomach prevents it from rupturing. The esophagus, in the thoracic cavity, a place of normally negative pressure, has no great positive pressure to surround it during emesis. If vomiting is too rapid to permit free egress of vomitus, or if any portion of the esophagus should go into physiological spasm during vomiting, that segment below it would be subjected to relatively high pressure.

Additional evidence of the spontaneity is afforded by reported cases of esophageal rupture occurring as a result of having been struck over the abdomen by a falling mantel,[12] run across the abdomen by a motorcycle,[13] or having been pinned beneath an automobile,[14] and having the esophagus ruptured by compressed air.[9]

Diagnosis: Very little of significance in the clinical history has been added since the original description by Boerhaave.[1] A close study of the

vast majority of the cases will reveal a similar clinical story. Usually the patient is a healthy man, often of stocky build, between 35 and 55 years of age. For any number of reasons, severe vomiting ensues, and at the height of a particularly strong emesis, the patient notes extreme upper abdominal and chest pain. The latter is most often noted on the left side, and may extend into the left shoulder. A sensation of "bursting something" in the chest is frequently noted. Shock, with clammy cold skin, rapid respirations, and cyanosis may promptly ensue. Emphysema of the cervical tissue may develop. Pain is unrelieved by the usual doses of narcotics. If nothing is done therapeutically, death almost invariably takes place within 48 hours, more often within the first 24 hours. Often, the diagnosis can be made from the clinical history.

This catastrophe is most frequently confused with perforated peptic ulcer of the stomach or duodenum, dissecting aortic aneurysm, acute hemorrhagic pancreatitis, coronary occlusion, and spontaneous pneumothorax.

In spontaneous pneumothorax, the accumulation of pleural fluid is slow and small in amount as contrasted to the accumulation of pleural fluid from irritation and spillage of gastric content from ruptured esophagus. Except for the occasional development of tension pneumothorax, the person with spontaneous pneumothorax is seldom very ill.

An upright or lateral decubitus roentgenogram of the abdomen will help rule out a perforated peptic ulcer and a normal serum amylase determination will generally exclude acute pancreatitis. An electrocardiogram will usually be abnormal if coronary occlusion is present.

The diagnosis of spontaneous esophageal rupture can be confirmed by the administration of lipiodol by mouth followed by a roentgenogram of the chest. If the esophagus be perforated, the dye will accumulate outside the esophagus, either in the mediastinum, or in the pleural cavity. Methylene blue in dilute solution may be administered by mouth and recovered by thoracentesis if the esophagus is ruptured. Often the pleural fluid will test acid to litmus paper due to the content of hydrochloric acid from the gastric juice.

Treatment: Antibiotics, blood transfusions, general supportive measures, oxygen therapy, and intratracheal anesthesia have all been important factors in survival and recovery. Treatment largely depends on the rapidity with which the diagnosis can be made. Sampson[15] feels that it is the rare patient that survives long enough for infection to develop. He advocates early thoracotomy with repair of the linear rent as soon as possible. Certainly, this is the optimum treatment if the diagnosis has been made early.

The great majority of esophageal rents that have been repaired later than 12 hours after onset of the tear have subsequently broken down with development of empyema. On the other hand, there are a number of patients who have survived following rib resection and drainage of the pleural cavity. It has been necessary to employ rather rigorous supportive measures in such individuals but the esophageal rent has healed spontaneously in such cases.

It would seem advantageous to employ immediate thoracotomy and repair of the esophageal rent if the diagnosis is made early. If the diagnosis is not made prior to 12 hours after the accident, it would seem more prudent to employ drainage of the pleural cavity, either through intercostal tubes or by means of rib resection. Later, when the esophageal rent closes spontaneously, decortication and reexpansion of the lung may be carried out.

A useful procedure to tide such patients over the long stage of malnutrition while the esophageal rent is healing is the performance of jejunostomy as advocated by Lynch.[10] Occasionally one is able to avoid jejunostomy by inserting a Miller-Abbott tube through the oral cavity, past the rent, into and through the stomach and duodenum, and permitting the tip to remain in the jejunum. One can then give jejunostomy feedings for an indefinite period of time.

Case Report

The patient was a white male, age 40, complaining of difficulty in breathing, and severe pain in the left chest and epigastrium. He had undergone repair of a right inguinal hernia on February 16, 1954 at the Adams County Hospital in Decatur, Indiana, and the operation had been carried out uneventfully under spinal anesthesia.

Later that day, he had a few sips of water and tea, when suddenly he became nauseated and vomited in a most forceful fashion. At that moment, he seemed to twist his body and immediately, he noted excruciating left chest and upper abdominal pain. Breathing became labored and extremely painful. Cyanosis of the nail beds was evident. His past history was essentially negative and he had never experienced dyspepsia or dysphagia.

Examination disclosed an acutely ill male breathing with difficulty and slightly cyanotic. The blood pressure was 140/80 mm. Hg., the pulse 130, and respirations 40 per minute. The breath sounds were absent over the left side of the chest and there was hyperresonance to percussion over the entire left side of the thorax. The apical beat was shifted to the parasternal line on the left side. A diagnosis of spontaneous pneumothorax was considered and a needle was inserted into the left pleural space. Free air was obtained but his condition failed to improve.

FIGURE 1

In the subsequent two days, serum amylase determination was normal and an upright roentgenogram of the abdomen failed to disclose free air under the diaphragm. A film of the chest (Fig. 1) disclosed complete collapse of the left lung with a large amount of fluid in the left pleural cavity. Methylene blue solution was administered orally and was recovered from the left pleural cavity, thus proving the diagnosis of spontaneous esophageal rupture.

On February 19, 1953, he was transferred to the Ft. Wayne Lutheran Hospital (No. 53-1408). He was obviously quite ill and it was necessary for him to be in an oxygen tent because of the difficult and labored respirations and the cyanotic tinge to the nail beds. It was impossible to control the chest pain, despite the administration of large doses of morphine. Two intercostal catheters were inserted into the left pleural cavity on the day of admission. He was placed on intravenous fluids, blood transfusions were given, and huge doses of antibiotics were administered. Oral intake was discontinued.

Copious quantities of thick yellow pus continued to drain from the intercostal catheters and on February 26, 1953, another larger catheter was inserted into the left pleural cavity and connected to mild suction. The following day, a Miller-Abbott tube was placed through the mouth, esophagus, stomach, duodenum, and into the jejunum and a 4,000 calorie jejunostomy formula was administered daily thereafter until the esophagus healed. On March 5, 1953, under general anesthesia, a rib resection was carried out. The left lung was completely collapsed and covered with a tough rind. The problem of pleural drainage was thus solved and his condition began to improve rapidly.

By March 21, 1953, it no longer was possible to recover orally administered fluids from the left chest, and accordingly the Miller-Abbott tube was removed and liquids were administered by mouth. He was dismissed from the hospital.

Two weeks later he was readmitted and under general anesthesia, decortication of the left lung and chest wall was performed. The site of previous rib resection was closed as was the operative incision, and two catheters were placed in the pleural space and connected to negative pressure. Supportive therapy and large quantities of antibiotics were given. He made an uneventful recovery and was dismissed on April 25, 1953. The wounds were healed and the left lung was completely expanded.

He has returned to his regular occupation and his duties and abilities have not been limited. The latest chest x-ray films show an essentially normal chest except for the absent ribs at the site of operative interventions.

SUMMARY

A brief review of the pertinent history, site of rupture, mechanism of rupture, diagnosis, differential diagnosis, and therapy of spontaneous rupture of the esophageal is presented.

A case of spontaneous esophageal rupture successfully treated by drainage of the pleural cavity followed by decortication of the lung and chest wall is prescribed.

RESUMEN

Se presenta un resumen de la historia pertinente, el sitio de ruptura, el mecanismo de ésta, diagnóstico, la diferenciación, y el tratamiento de ruptura espontánea del esófago.

Se describe un caso de ruptura espontánea del esófago tratada con éxito por drenaje de la cavidad pleural seguido de decorticación del pulmón y de la pared de tórax.

RESUME

Les auteurs font une revue rapide de l'historique de la rupture spontanée de l'oesophage. Ils rappellent le siège de la rupture, son mécanisme, les éléments du diagnostic différentiel, et du traitement. Ils rapportent une observation de rupture spontanée de l'oesophage heureusement traitée par drainage de la cavité pleurale, suivi de décortication du poumon et de la paroi thoracique.

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Multiple Saccular Aneurysms of the Thoracic Aorta with Spontaneous Rupture into the Esophagus: Report of a Case*

ADRIAN G. KRUDY, M.D., Cleveland, Ohio, and
JOSEPH A. SMITH, M.D., F.C.C.P., Glen Gardner, New Jersey

Saccular aneurysm is "a pouching of the vessel at one point" (W. Boyd[1]). Saccular aneurysms of the aorta are almost always syphilitic in origin. Because syphilitic aortitis usually develops in the thoracic aorta, it is the thoracic portion of this vessel which is especially affected. Syphilitic aneurysms of the abdominal aorta are relatively infrequent. Aneurysmal sacs may arise from the ascending, transverse, and descending segment of the arch, and, least frequently, from the descending aorta.

Multiple sacs may develop, though this is unusual. Of 633 cases of saccular aneurysms of the thoracic aorta, reported by Kampmeier,[2] only 23 or 3.6 per cent were multiple.

Thoracic aneurysms may rupture into the surrounding structures including the pericardium, pleural cavity, esophagus, bronchus, etc. The present report is concerned with a case of multiple saccular aneurysms of the thoracic aorta, one of which perforated into the esophagus.

*From the New Jersey Sanatorium For Chest Diseases, Glen Gardner, New Jersey.

Case Report

A 48-year-old white man was admitted to the New Jersey State Sanatorium, September 23, 1952, with the diagnosis of active far advanced bilateral pulmonary tuberculosis. There was previous sanatorium treatment elsewhere in 1941-1943. Six months prior to this admission he developed productive cough and marked weight loss and fatigue. He gave a history of hospitalization for bleeding peptic ulcer three years ago. Two years ago he was given antisyphilitic treatment of daily intramuscular injections for a period of two weeks. He admitted excessive alcoholic intake for several years. He had worked in a zinc mine for 18 years and in a coal mine for two years.

He was a well-developed, thin, pale, white man appearing to be chronically ill, nervous and apprehensive. His temperature was 99.0 F. Blood pressure was 150/85 mm. Hg. and the pulse rate 100 beats per minute, with regular sinus rhythm. A systolic murmur was audible at the aortic area. Auscultation revealed diminution of breath sounds over both apical regions. The liver, kidneys, and spleen were not palpable. The pupils were equal, regular, and reacted normally to light. The knee jerks were present. There was a coarse tremor of the extended fingers. The red blood cell count was 4,130,000 per cubic millimeter, with 13.0 gm. of hemoglobin per 100 cc. The white blood cell count was 10,600 per cubic millimeter, with a normal differential count. Blood sedimentation rate was 28 millimeter per hour. Results of routine urinalysis were essentially normal. Sputum contained acid fast bacilli. The blood Mazzini reaction was repeatedly positive in dilutions through 1-32. Examination of the cerebrospinal fluid showed the total protein to be 32 mgms. per 100 cc. Kolmer test of spinal fluid was negative. The chest x-ray film showed a mottled type of infiltration in both upper lung fields, and there was widening of the upper mediastinum (Fig. 1). X-ray film and fluoroscopic studies after barium swallowing were suggestive of an aneurysm of the aortic arch.

Two days after admission he became restless and disoriented, particularly at night. This was thought to be due to the abrupt cessation of alcoholic intake. Sedatives resulted in improvement in three days. Treatment of the lung condition consisted of bed rest and of daily administration of 1 gm. dihydrostreptomycin intramuscularly, combined with 12 gms. of para-aminosalicylic acid orally, for five months. There was clinical improvement with diminution of cough and weight gain, however, his sputum remained positive and there was but little change in the x-ray film findings. On April 6, 1953, he suddenly vomited bright red blood, amounting to about 300 cc. Because of the history of peptic ulcer, he was treated with milk and cream diet, aluminium hydroxide, tincture of belladonna, and sedatives. The next day he had

FIGURE 1: Roentgenogram showing widening of the upper mediastinum. Also there is a mottled type of infiltration in both upper lung fields.

recurrence of the hematemesis, with the loss of another 300 cc. of blood. Blood transfusion was given and he appeared improved. On the evening of April 10, 1953, he became nauseated and again vomited bright red blood-clots. He also complained of substernal pain. The next morning, he went into shock and died.

At necropsy the thoracic aorta showed the gross features of syphilitic aortitis, and on the descending arch were the orifices of three saccular aneurysms (Fig. 2), two of which were quite small, their mouths measured 0.7 by 1.0 cm., the sacs were 3.0 by 1.5 cm. and 1.5 by 1.0 cm., respectively. The mouth of the third and largest one measured 1.2 by 1.0 cm. The sac was 4 cm. vertically, 3 cm. transversely, and 2.5 cm. ventro-dorsally, it directed posteriorly, was adherent to the left anterior wall of the esophagus, and perforated into its lumen with a small irregular opening (Fig. 3). The stomach, duodenum, and the small intestines were filled with clotted blood. There was no evidence of ulceration of the gastric or duodenal mucosa. The upper part of each lung was firmly attached to the chest wall and on cut section showed the picture of chronic fibroid tuberculosis. Microscopic examination of the thoracic aorta showed marked sclerotic changes in the subintimal region with thinning and there were areas of dense perivascular lymphocytic infiltration in the adventitia, with vascularization of the media.

Discussion

This case illustrates that the diagnosis of aortic perforation into the esophagus should be considered in a patient with repeated hematemesis, when aneurysm of the thoracic aorta is also demonstrated.

A case of aortico-esophageal perforation with fatal hematemesis was reported recently by Calenda and Uricchio[3] who state that rupture of an

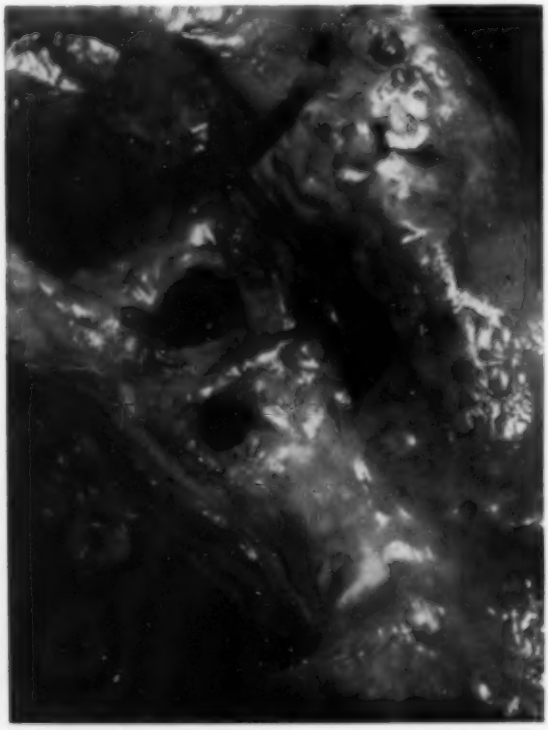

FIGURE 2: Necropsy specimen showing the three aneurysmal orifices on the descending aortic arch. Indicator is in the largest aneurysm which has ruptured into the esophagus.

aortic aneurysm into the gastrointestinal tract is "extremely rare." "Perforation of the thoracic aneurysm into the esophagus is even more unusual, since in the past 27 years only five such cases have been described in the world literature." However, review of earlier studies of large series of thoracic aneurysms showed that rupture into the esophagus was not a rare occurrence. Lucke and Rea,[4] in their series of 263 aortic aneurysms (including also fusiform and dissecting forms), found that 233 were located in the thoracic portion. Rupture of the sac took place in 51 cases; of these, 5 (9.8 per cent) perforated into the esophagus. Boyd[5] made a study of 4000 aneurysms of the thoracic aorta and found that 1197 patients died of rupture into various organs, and of these, 112 (9.4 per cent) ruptured into the esophagus. Of the 633 cases of saccular aneurysms of the thoracic aorta, reported by Kampmeier,[2] rupture of the sac occurred in 98 cases. The most common site of the rupture was into the esophagus with 20 cases (20.4 per cent) (19 ruptured into the esophagus alone and one to the esophagus and the left pleura).

The scarcity of reported cases in the recent literature must be due to the decreased incidence of syphilitic aneurysms. Kampmeier[2] analyzed 591 cases of thoracic aneurysms in 5 year periods from 1906 to 1935 in-

FIGURE 3: Necropsy specimen, posterior view. Indicator is placed through the perforation from the largest aortic aneurysm into the opening on the wall of esophagus. The aneurysmal sac is cut open showing laminated blood-clots.

clusive, as related to hospital admissions (Charity Hospital, New Orleans), and found a decrease in frequency by more than half. This decrease is probably due to the more efficient treatment of early syphilis since the introduction of arsenotherapy (Ehrlich, 1910) and bismuth compounds (Sazerac and Lævaditi, 1920). Since aortic aneurysms are usually not evident for 10 to 20 years after infection in untreated individuals, the effect of the more efficient treatment was not shown until the late 20's and early 30's. In recent years penicillin has greatly simplified the treatment of syphilis, and it is hoped that, with a more effective syphilis control program and the more widespread use of modern syphilotherapy, syphilitic aneurysm of the aorta will become a medical rarity.

SUMMARY

A case of multiple saccular aneurysms of the thoracic aorta, with spontaneous rupture into the esophagus causing fatal gastrointestinal hemorrhage, is presented.

RESUMEN

Se presenta un caso de aneurismas saculares mútiples de la aorta torácica con ruptura espontánea dentro del esófago y hemorragia fatal.

RESUME

Les auteurs rapportent un cas de poches anévrysmales multiples de l'aorte thoracique. Il en est résulté une rupture spontanée dans l'oesophage ayant déterminé une hémorragie gastrointestinale mortelle.

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Editorial

Doctors and Peace

The longing for peace is ever present in the human heart. It was felt by President George Washington when he said that his first wish was that there would be peace among all people and that the only contention should be who best could serve his fellow man.

This spirit was in evidence at the Interim Meeting of the American College of Chest Physicians in Miami Beach when doctors talked about the warm hospitality and friendliness of our colleagues at the meetings held in other lands. As one man expressed it "There would be no war if the decision rested with the doctors of the world."

The medical profession has a long record of idealism—a sense of values which has made the good physician dedicate himself to the saving of human life and not its destruction. It is epitomized in the Oath of Hippocrates which is recognized by doctors throughout the world as the medical professions ideal in our relationship to our fellow men. Is it strange then that physicians throughout the world should have a natural bond of sympathy and understanding toward each other? It is well illustrated by the doctor's conduct in war. His enemy is not the human foe but rather the disease and body mutilation that comes to the soldier be he friend or enemy. You may see the doctor administering his skill to the patient perhaps oblivious to the make of his uniform.

It is told that during the final hours of the Berlin siege of World War II a German surgeon confined to the basement of his bombed hospital was suturing the wounds of one of the many casualties surrounding him. The enemy entered. A Russian medical officer about to take charge asked "who is operating?" Someone answered "Professor Sauerbruck." Without hesitation the medical officer who had known of Professor Sauerbruck walked up and saluted him. No bitternesss, no vengeance nor hate there. They spoke the same healing language. They understood.

The healing hand of medicine knows no boundaries, no creeds, no race nor color. Left to itself without the grasping hand of political dictatorship it will rise to its noble station in life asking for nothing more than the freedom of struggle to see who best can serve in the preservation and enrichment of mankind.

Medicine and music both speak this universal language of peace and good will. If they ruled the world there could be no war.

ARNOLD S. ANDERSON, M.D., F.C.C.P.

Teaching Chest Disease*

The Student as an Individual

DAVID G. GREENE, M.D.**

In 1910 the report of Dr. Abraham Flexner called attention to the chaotic situation which then existed in the field of medical education in the United States. Diploma mills and proprietary institutions were only slightly worse than some departments of even the best medical schools. Few medical diplomas carried with them the assurance that the recipient had met acceptable requirements in the various branches of medicine.

In the intervening 45 years a great deal of effort has been directed toward raising the *standards* of medical education in this country. This has been accomplished to the extent that a diploma from an American medical school now indicates that a student has attained a reasonable acquaintance with a rather specific body of knowledge and some proficiency in a common set of technics. A large number of medical students are now taking, and for the most part passing, examinations given by a national board. Much of this *standardization* has been beneficial in raising the level of medical practice. But has it also had some deleterious effects? Have we standardized down as well as standardizing up? Do the present standards prevent bold and original thinking on the part of medical students and medical faculties? To each of these questions I believe the answer is yes.

One false premise of some advocates of standardization is the consideration of medical students as standard college graduates taking a standard program designed to make them standard doctors. Since no such thing as the standard college graduate, medical student or doctor exists, the medical schools run the risk of directing their activities toward hypothetical persons. This is not an idle sophistry. It is possible and perhaps preferable although more difficult to direct medical education toward the needs of the individual student.

The simplest method of doing this, and one which has worked well in schools where it has been given some trial, is to allow the student greater choice in the selection of his course of study. This plan does not imply the offer of a completely cafeteria-style course of study. Rather it suggests that the required courses be cut so drastically that they can be presented in about one-half the time of four academic years. To the critic who protests that the minimum essentials "cannot be covered in such time," I reply that they cannot be covered in the currently alloted time if one means by "covering" attaining an intelligent and useful understanding of the principles of the basic sciences and the clinical disciplines. The fields are too large. If one means by "covering" the transcription by students

*The fifth in a series of articles prepared under the sponsorship of The Council on Undergraduate Education of the American College of Chest Physicians.

**From the Department of Medicine and Physiology of the University of Buffalo School of Medicine, and the Buffalo General Hospital.

of the professor's lecture notes, then half the present time is too much to allot to such a sterile process. One is then faced with the problem of what can be omitted. At present from a heavy intellectual meal the student rejects or neglects the items least likely to aid him in his immediate goals—passing examinations and getting a diploma. These same rejected items may be the very ones on which interest would prompt him to concentrate, but the system does not allow for that. How often have medical students complained of the lack of time to digress from the assigned course material to study fascinating footnotes connected with last year's or next year's work. Interest is an essential of real learning. But the present system discourages this easiest and best type of learning. Ideal education consists of arousing a student's curiosity and then allowing him enough discretion, time and facilities so that he can concentrate on fields which appeal to him.

Ah, but such a whimsical way of doing things will never do—how about the general practitioner and the country doctor who must be prepared to treat diseases of the skin and its contents? It won't do for them to know all about the ears and nothing about the eyes. Quite so, and men heading for such careers should certainly diversify. More than that, they should seek opportunities to "specialize" in preparation for what is in fact a specialized career. The Academy of General Practice recognizes this with its current emphasis on continuing education.

Curriculum change is the lesser part of the problem. More important is the emphasis on the individual consideration of the individual student. This was brought home to me on my first day of medical school, in the office of the dean, which I had visited once briefly 10 months before. I walked in expecting to explain that I was a new student, and half fearing that when I finally got to the right official some defect in my credentials would be discovered. I still remember vividly my surprise and delight at being greeted warmly by name before I could introduce myself. This personal relationship of student with faculty was preserved outstandingly during my student career by our professor of physical diagnosis, Dr. Henry Jackson Jr. During his course he became personally acquainted with 125 students, and gave the impression to each that this was a particularly valued friendship. Individual selection of courses and personal contacts with faculty members are then two means of individualizing medical education. As physicians we individualize treatment of our patients. The same principles apply equally to our relations with our students.

A corollary of the individualization of the curriculum and of the faculty-student relationship is the transfer to the student of a large part of the responsibility for his own education. Many teachers find this a difficult transfer to make. A prevalent faculty characteristic is the desire to saturate a student with everything known on a subject—to "tell all." This process satisfies the conscience of the teacher, and swamps the student. What is needed is an admixture of restraint on the part of the faculty and the courage to allow the student to make mistakes.

The usual objection raised to this line of thought is that the student is in no position to know "what is important." One flippant answer is to admit that in this rapidly changing world of science neither does anyone else. A more serious reply is that no one is more anxious to learn what is important than the medical student. He will find the heart of the problem regardless of the manner in which it is presented, and his own synthesis may be more valid for him than one of a previous generation. The student is the best authority on where the gaps are in his own training, and no one knows better than he where his true interests lie. Moreover, his curiosity may lead him into unexplored paths undreamed of by his elders. Louis Pasteur after all was a crystallographer who observed some extraneous matters which led him to an entirely new science, bacteriology. It is important to allow young men to follow exciting leads which are unrelated to course credits. Now, suppose that the interests of an individual carry him quite beyond the accepted borders of medicine into unexplored territory. Is this to be regarded as the loss to the fold of one errant sheep, or a gain to the community of one original thinker? Perhaps this is a trend we should foster. Some may fear that such self-determination run riot will surely undermine the supply of physicians of the usual mold. Unfortunately the danger is quite the other way; even with large degrees of freedom the usual medical student sticks to the usual path. However free the curriculum, licensing examinations will prevent all but the most venturesome from more than short excursions into the byways. And that is another story.

* * * * * *

The College of Chest Physicians has avoided the temptation to try to standardize the teaching of chest disease in medical schools. With the pressure of curriculum changes the tendency might well be to try to teach more and more in less and less time, and to crystallize the material presented, a self-defeating process. Rather than promoting a standard prospectus, the College has served progress in chest disease better by stimulating imaginative teaching which will attract the enthusiastic student.

"HOLD THE TORCH HIGH"

The Founders of the American College of Chest Physicians built well when they dedicated themselves to the cause of spreading knowledge and information concerning diseases of the chest. It takes more than a mere statement of purpose for a group of men to attain a desired goal. The group must work individually and collectively toward the attainment of the desired end. This unity of effort on the part of the Fellows of the College has been conspicuous throughout the life of the College.

There have been differences of opinions at times, between individuals, as to the most desirable procedure to be followed. These differences have been resolved through discussion to the end that the will of the majority has always prevailed. I am moved to speak of this, at this time, because it would be so easy to create confusion if the chosen officers of responsibility should ever permit any deviation from this policy.

With the lapse of time and through increasing experiences, the Fellows have found it desirable to establish numerous councils and committees, each composed of Fellows selected because of their particular skills in their chosen fields of endeavor. Many far-reaching contributions have resulted through the efforts of these groups. The prerogative of the individual to speak and act as an individual has never been lost but, at the same time, the groups have reported on the basis of the majority opinion.

New developments and increasing experience may have led to a change of recommendations but these changes have been adopted only after very careful considerations by responsible individuals.

I would urge all the Fellows to give of their time and effort toward the continued effectiveness of the various councils and committees. The high standards set for the Chapter and Annual Meetings, and of the International Congresses, should never be lowered. Meetings with formal and informal exchange of information is a time-honored and proven method of spreading knowledge and of creating understanding and good will between individuals and between peoples.

The Fellows cannot afford to permit the College to become involved in matters that are not directly concerned with their primary goal. Mutual interest in information and knowledge of diseases of the chest with service to humanity is the source of our "Common Bond."

The American College of Chest Physicians will continue to rank high among medical organizations so long as the Fellows of the College continue in their avowed purpose of service to humanity through the physician.

Mrs. Hudson and I wish to express our deep appreciation to the Fellows of the American College of Chest Physicians and, to all the members of the councils and committees, local, national and international, for their unlimited cooperation which has meant so much to us in our feeble efforts for the good of the College. Our Executive Director and the Staff in the Home Office of the College have exhibited a brand of loyalty to the cause of the American College of Chest Physicians that is superb.

Mrs. Hudson and I will always cherish the memory of our many pleasant experiences of the past years and pledge our continued loyalty and support to the American College of Chest Physicians.

William A. Hudson

PRESIDENT

JAMES H. STYGALL
President
American College of Chest Physicians
1955—1956

DR. JAMES H. STYGALL
TAKES OFFICE AS COLLEGE PRESIDENT

At the Annual Presidents' Banquet held on Saturday, June 4, at the Ambassador Hotel, Atlantic City, in connection with the 21st Annual Meeting of the American College of Chest Physicians, Dr. James Henry Stygall of Indianapolis, Indiana was installed as President of the College for the year 1955-1956.

Dr. Stygall was born on March 10, 1887 and received his degree of Doctor of Medicine from the University of Buffalo Medical School in 1910. After graduation from medical school, he served on the staffs of several tuberculosis sanatoria, and in 1918, enlisted in the Army Medical Corps during World War I and received a commission as Captain. He was later commissioned as Major in the Reserve Corps.

Following World War I, Dr. Stygall was appointed Superintendent of the Rocky Crest Sanatorium in Olean, New York. In 1921, he accepted the position of Medical Director of the Indiana Tuberculosis Association, during which time he established tuberculosis clinics throughout Indiana. He was elected President of the Indiana Tuberculosis Association in 1928.

Dr. Stygall is a charter member of the American College of Chest Physicians and served as Chairman of the Board of Regents of the College for two years. He also served as Chairman of the Council on Tuberculosis Committees for a number of years and aided in the establishment of committees on tuberculosis in state and county medical societies throughout the country. Dr. Stygall served as chest consultant to the Selective Service System during World War II and has been a member of the Board of Managers for the Indiana State Sanatoria for many years.

At the present time Dr. Stygall is Chief of Staff of the Flower Mission Hospital, which position he has held for a number of years, is Chairman of the Tuberculosis Committee of the Indiana State Medical Association, and Controller of Tuberculosis for the city of Indianapolis and Marion County. He has taught medical students for 30 years and is Assistant Professor of Medicine at the Indiana University Medical School.

Dr. Stygall is a Fellow of the American College of Physicians and the American Medical Association. He holds membership in numerous other medical and fraternal societies.

Dr. Stygall and his wife Della reside in Indianapolis. They have one married son, James Jr., and a granddaughter.

Obituary

SIR ALEXANDER FLEMING

D.Sc., M.B., F.R.C.P., F.R.C.S., F.R.S., Regent for Great Britain

1881-1955

Sir Alexander Fleming died suddenly of a heart attack in London on March 11th, 1955. He was seventy-three.

As his father was a farmer in Ayrshire, Scotland, Fleming received his early education at the local village school and the nearby Kilmarnock Academy until the age of thirteen. He then went to live with a brother, already practising as a doctor in London, and after continuing his education at the Polytechnic Institute, became a shipping clerk. On the advice of his brother he decided, however, to study medicine at St. Mary's Hospital Medical School. His ability was immediately evident. He went on from taking the senior entrance scholarship in natural science to almost every class prize and scholarship as a student, and graduated M.B., B.S. with honors and a gold medal in 1908. Within a year he had become a Fellow of the Royal College of Surgeons.

His real interest from the beginning was in bacteriology. In 1912 he became an assistant to the famous Sir Almroth Wright and returned to him as a lecturer at St. Mary's Hospital at the end of full service with the R.A.M.C. as a captain during the First World War. In 1928 he was elected professor of bacteriology, and held this post until 1948, when he was given the title of Emeritus Professor, as head of the Wright-Fleming Institute of Micro-Biology. By 1922 he had made his first notable discovery, lysozyme.

It was typical of Fleming's unassuming attitude to all we owe him that he continued to the end to claim that his discovery of penicillin in 1928 was a mere accident. We know, however, that only hard work, a trained brain and a discerning eye saw the possibilities in the original mould which, by chance contamination of a culture plate left lying on his work-bench for a week had destroyed adjoining colonies of staphylococci. Again it was typical of him that he continued working on this mould, finding that the crude fluid in which it grew was strongly antibacterial and non-toxic, but unfortunately in crude form too unstable for injection purposes in human-beings. Yet he was able through his researches to suggest that it might be of definite use for the treatment of infected wounds. Nowadays we realize that his original and world famous paper in 1929 on what he had called penicillin was indeed a remarkable publication; it covered most of the outstanding difficulties and outlined what he had done in attempts to clarify them. Although it remained for Florey and his co-workers to bring penicillin to practical use, we do not forget that it is to Fleming working alone that we owe its discovery.

He received innumerable honors, but remained the same rather taciturn Scot, with the same unassuming manner. A Knighthood was followed by the Fellowship of the Royal Society, Fellowships of both Royal Colleges of Physicians, and nine honorary degrees of Doctor of Science, three of them from American universities. He was a Fellow of the Royal Society of Copenhagen, a Foreign Associate of the French Academy of Medicine and a Commander of the Legion of Honor. He was appointed to many Lectureships, such as the Cutter at Harvard University, the Mayo Foundation at the University of Minnesota, and the Linacre at Cambridge University. He was awarded the Gold Medal in Therapeutics of the Society of Apothecaries, and jointly with Sir Howard Florey, the Gold Medal of the Royal Society of Medicine.

In 1950 he received the Gold Medal of the American College of Chest Physicians at its First International Congress in Rome, and from that year until his death was Regent for Great Britain.

His ashes lie in the Crypt of St. Paul's Cathedral. Their deposition there on March 18th was a fitting last testimony to what Britain, and the world, owes to Alexander Fleming.

RICHARD R. TRAIL, M.D.
Governor for England

BOOK REVIEWS

DISEASES TRANSMITTED FROM ANIMALS TO MAN. Fourth Edition. By Thomas G. Hull, Ph.D. Secretary, Council on Scientific Assembly, Director, Bureau of Exhibits, American Medical Association. Charles C Thomas, Springfield, Ill., 1955. Pp. 717, price $12.50.

Thomas Hull and his expert contributors, of whom there are 24, have done a fine service in keeping this book revised so it has always contained the latest and most authentic information. The present fourth edition is composed of 717 pages with 104 illustrations and 66 tables. The work of some 1200 authors has been drawn upon in the preparation of this volume.

There are more than 100 diseases transmissible from animals to man which are recorded in this volume with accurate documentary evidence, and the salient facts that are known about them. Chapter 1, by William H. Feldman, is an excellent presentation of tuberculosis transmissible from animals to man. The various types of tubercle bacilli from different animal hosts are discussed and it is pointed out that from a public health point of view, tuberculous cattle constitute the most important animal source for tubercle bacilli that are virulent for man. Although in the United States bovine tuberculosis has been reduced to a remarkably low level, he says, "Yet so long as a single focus of infection remains, there exist the essentials necessary for the dissemination of the infection. The attack against the tubercle bacillus must be prosecuted with vigor and without compromise. An attitude of complacency, or one in which satisfaction with past achievements takes precedence over the importance of the task yet to be done will defeat any plan of approach, no matter how adequate it may appear to be." Feldman further points out that the remarkable achievements of the veterinary profession in practically eliminating tuberculosis in cattle in the United States constitutes a notable example for the guidance of physicians who are concerned with eliminating tuberculosis in human beings. He says, "The problem must be attacked nationally and with vigor, with the thought ever in mind that tuberculosis in a highly contagious disease."

Excellent accounts of anthrax, brucellosis, glanders, rabies, tularemia, and a vast number of others now known to be transmissible from animals to man are recorded in the remaining 37 chapters.

Every veterinarian, physician, health officer and other professional health workers should have this book available. Moreover, it should be in every public library, as it gives the general reader facts about the magnitude of this problem, and the importance of public support everywhere in the control and eradication of these diseases.

J. A. Myers, M.D.

BASES OF RADIOLOGIC INTERPRETATION; RADIOGEOMETRY. Two Volumes, 524 pages, 683 figures and diagrams. Masson et Cie., Paris, 1954.

These two volumes propose a radiogeometric approach to radiologic interpretation. The first volume consists largely of a discussion, whereas the second volume forms an atlas type of presentation of figures and diagrams. The effect of absorption and transmission of roentgen rays as affected by linear, triangular, circular and other forms of densities is presented in order to attempt to show how these influence the final roentgenographic result. Several geometric theories are discussed and are amply illustrated in the atlas. The geometric principles involved in tomography are also included. In the latter portion of the text and of the atlas, the correlation of these geometric principles with the roentgenographic studies of various structures of the bodies is undertaken. Discussion of the various structures such as the heart, great vessels, skull, ventricular system and other organs and tissues represented in this fashion.

Charles M. Nice, M.D.

DIAGNOSIS OF BRONCHOGENIC CARCINOMA (La Diagnosi del Carcinoma Broncogeno), by Aldo Biobbi, M.D., Diagnostic Center for Pulmonary Tuberculosis, Milan, Italy. Published by Edizioni Minerva Medica, Turin, Italy. October, 1953.

The purpose of this monograph is to present a didatic and complete analysis of the problems confronting the clinician in diagnosing bronchogenic carcinoma.

The author has been particularly interested in reporting and correlating contributions from European and American literature. He also presents an analytic study of 219 cases of primary carcinoma of the lung observed at the diagnostic center for tuberculosis in Milan from 1948 to 1953. The increasing incidence of death due to primary carcinomas of the lung in this large industrial city (pop. 1,250,000) is well illustrated by the following figures. In 1952, 204 out of 1791 deaths due to malignant tumors were caused by primary carcinomas of the trachea, bronchi and lungs (0.16%). The number of deaths due to pulmonary tuberculosis in the same period were 281 (0.22%).

In Dr. Biobbi's series of 219 cases diagnosed over a period of over 5 years, 95.4% were males and 4.6 females. Six cases were under 40 years of age (2.7%). 199 cases between the age of 40 and 60 (85.9%). 122 (55.7%) of the patients were living in Milan and 97 (44.3%) in rural areas. Only 70 (31.3%) were heavy smokers (20 cig. daily). Histologically 59.8% were classified as squamous cell carcinomas, 33.3% as oat cell carcinomas and 6.9% as adenocarcinomas. Other figures reported the incidence of cough, pain, physical signs, bronchoscopic findings and incidence of incorrect clinical diagnosis.

The chapter headings indicate the scope of the work: Frequency, pathologic anatomy, symptomatology, physical diagnosis, radiologic diagnosis, evolution and therapeutic criteria.

The importance of early diagnosis and the necessity of team work with cooperation of the clinician, the radiologist, the surgeon and the pathologists, is emphasized by the author.

This work is profusely illustrated with excellent x-ray, reproductions and technically outstanding tomograms and contains a rich bibliography of 110 European and American contributions. It is well organized, very comprehensive and will prove to be very useful to chest specialists, general practitioners, and to any physicians interested in the problem of early diagnosis of carcinoma of the lung.

S. M. Farber, M.D.

ANNOUNCEMENT

The Second International Congress of Allergology will be held in Rio de Janeiro, Brazil, November 6-13, 1955, under the chairmanship of Dr. F. W. Wittich, President of the International Association of Allergology. A wide and extensive program dealing with almost all important problems of allergy and related immunology, biochemistry, pharmacology, and therapeutics, is being prepared by the International Association of Allergology and the Brazilian Allergy Society. Physicians wishing to present papers should send their requests to Dr. Wittich, 424 La Salle Medical Building, Minneapolis 2, Minnesota. Information concerning the Congress is available from Dr. Bernard N. Halpern, 197 Boulevard Saint Germain, Paris, France.

THE INDEX

TO THIS VOLUME HAS BEEN REMOVED FROM THIS POSITION AND PLACED AT THE BEGINNING OF THE FILM FOR THE CONVENIENCE OF READERS.

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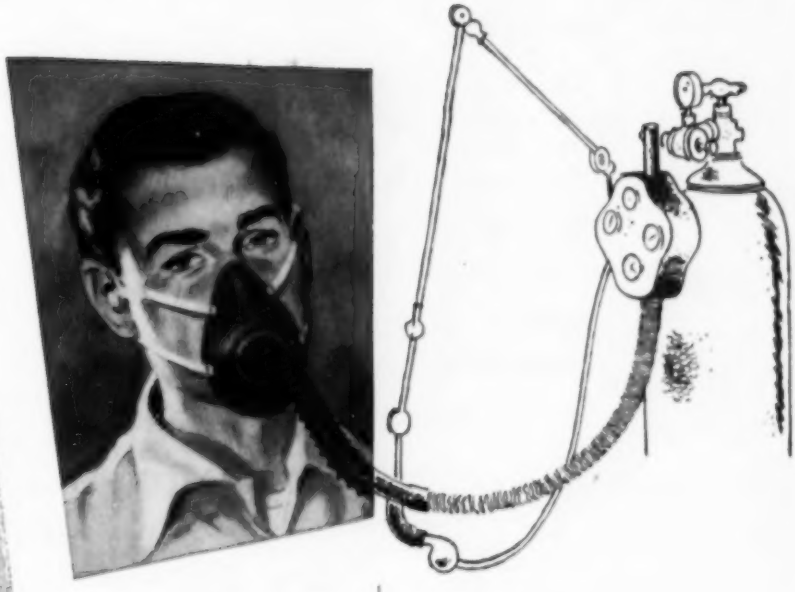

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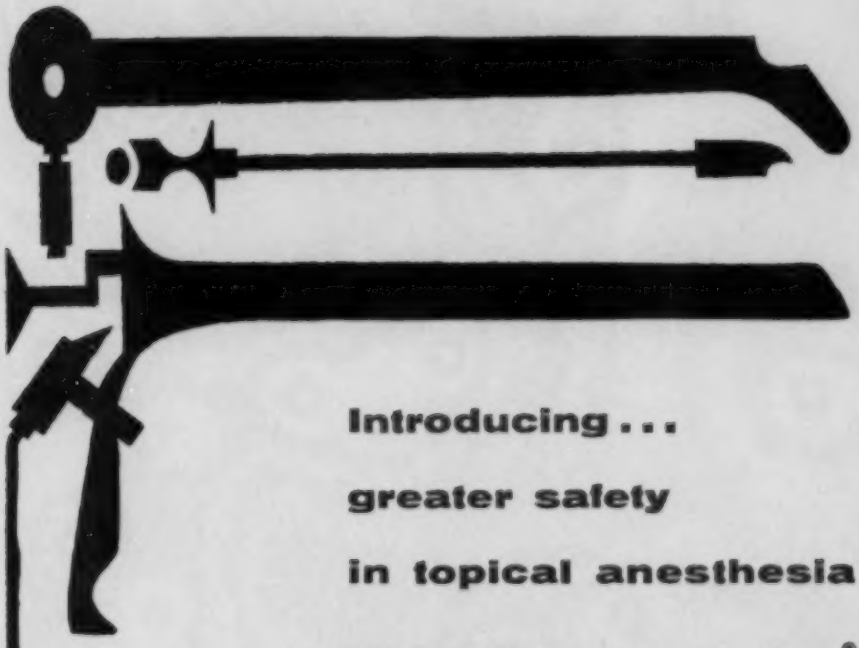

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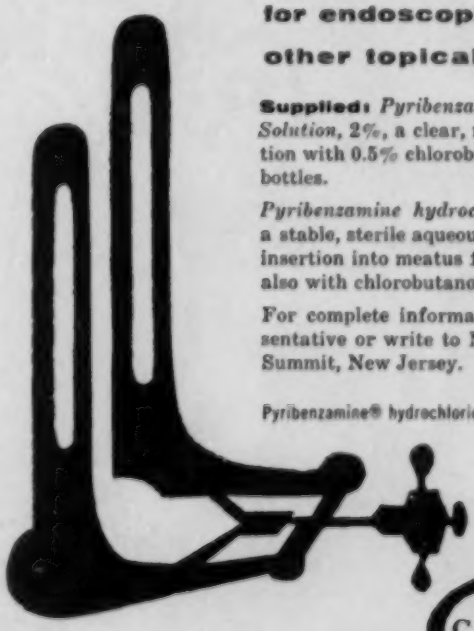

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